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# Portacaval Shunt in Patients with Cirrhosis

## After-Effects of the Operation

JOHN A. BENSON, JR., *M.D.*, Portland, Oregon

• **The subject of the indications for operation for portacaval shunt bristles with problems to which a satisfactory answer cannot yet be given. I have discussed those events which supervene after a portacaval shunt which, in the present state of knowledge, has been directed as proper and well-intentioned therapy. It is against the knowledge of such post-shunt events and the complexity of their management, as well as the success of the operation and the risk of mortality, that one must balance the indications for surgery. This is particularly pertinent to the application of prophylactic shunt surgery now under consideration.**

BEFORE DISCUSSING some of the empirical results in patients with chronic liver disease upon whom such shunting operation has been performed, I would like to enumerate some of the physiologic events which can be expected. If one creates in dogs a side-to-side anastomosis between the portal vein and the inferior vena cava below the liver (Figure 1A), the only impaired function which can be measured is that of regeneration of liver tissue after partial hepatectomy. If one then ligates the portal vein on the liver's side of the portacaval anastomosis (Figure 1B), thereby creating an Eck fistula, further changes ensue. (Such an Eck fistula is duplicated in humans, incidentally, by a standard end-to-side anastomosis (Figure 1C) between the portal vein and the inferior vena cava.) Animals that have had these operations undergo gradual atrophy of the liver. After some delay a progressive episodic stuporous condition, which has been called "meat intoxication," begins. This occurs whether or not meat is present in the diet and seems to be more clearly related to constipating diets. Protection of such dogs from the "meat intoxication" of an Eck fistula can be afforded by gradual occlusion of the portal vein below the fistula (Figure 1D). Complete recovery from the symptoms of Eck fistula is also effected if the vena cava above the portacaval anastomosis is occluded (Figure 1E). Bollman[2] speculated that the common denominator here is prevention of pulse waves from the right heart down through the anastomosis and portal system to the intestines.

Portacaval anastomosis halves the blood flow through the liver of animals.[3] Perhaps because the terminal parenchyma along the vascular system is quite hypoxic, tests of liver function show impairment after portacaval anastomosis. For example, the by-passed liver can then clear only one-third as much bromsulphalein each minute as the liver of a normal animal can. Furthermore, there is a decreased output of glucose by the liver after stimulation with insulin. Whipple,[14] using Eck fistula preparations, found that production of bile salts was

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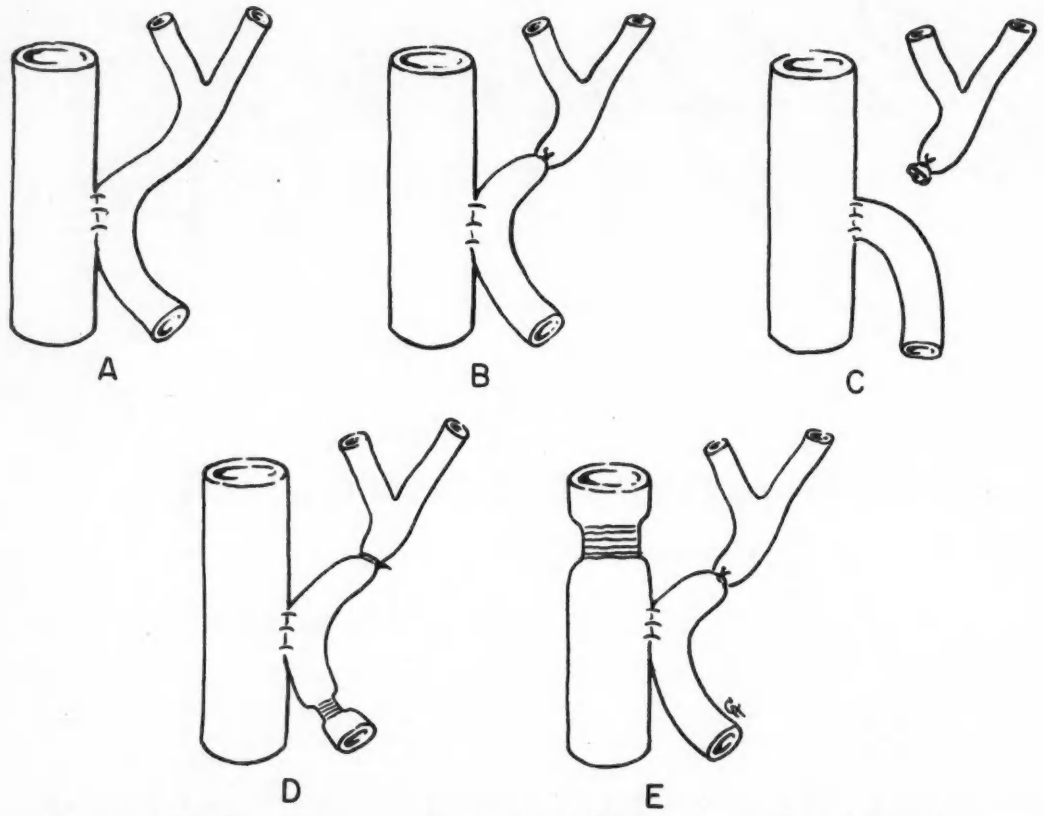


**Figure 1.—Various placements for portacaval anastomosis, the inferior vena cava just below the liver always appearing on the left: *A*. Side-to-side portacaval shunt. *B*. Side-to-side portacaval shunt with ligation of portal vein above the shunt, or an Eck fistula. *C*. End-to-side portacaval shunt. *D*. Eck fistula with occlusion of portal vein below the shunt by cellophane stricture. *E*. Eck fistula with occlusion of the inferior vena cava above the shunt by cellophane stricture.**

less than normal. There also were periods of a definite incapacity to utilize dietary protein in the formation of new hemoglobin and plasma proteins, and the utilization of ingested iron toward correction of a nutritional anemia was less efficient.

In dogs the ability of the remaining normal liver to regenerate after partial hepatectomy or injury by carbon tetrachloride is impaired by a portacaval anastomosis. Repair after partial hepatectomy is better if portacaval transposition is performed instead. Such a transposition perfuses the liver through the portal vein with caval blood from the kidneys and lower extremities. Liver regeneration is also improved by arterialization of the hepatic stub of the portal vein in an animal with an end-to-side portacaval shunt.[8] Such animals with total arterialization of the liver maintain body weight, have a greater survival and do not develop "meat intoxication," in contrast to those with Eck fistula. Thus it would seem that the ability of the liver to regenerate depends in part on adequate flow of blood through it.

The condition referred to as "meat intoxication" deserves further comment. In dogs, such episodic stuporous states have been observed with non-specific changes in electroencephalograms and eventually elevations of the blood ammonia. The episodes of intoxication occur at an accelerating pace, despite the administration of oxytetracycline or neomycin by mouth and irrespective of a limited oral intake of protein, until the animals die in 12 weeks or so, emaciated and comatose. Their brains show increased numbers of protoplasmic astrocytes.

### DIFFERENCES IN EXPERIENCE IN HUMANS

In considering the relationship of the foregoing facts to corresponding conditions in human beings, it must be borne in mind that in humans we are dealing not with a normal liver or hepatic circulation, but with one affected by chronic disease. End-to-side portacaval shunt in a human with cirrhosis of the liver is not regularly attended by either episodic stupor or relentless deterioration to early

death. This difference deserves emphasis. There are at least two possible reasons why a human with cirrhosis does better than a dog with an Eck fistula. First, the dog that has also had an occlusion of the portal vein (Figure 1D) with development of adequate collaterals, is protected from "meat intoxication" and death. A corollary of this exists in the human who before a shunting operation has acquired spontaneous collaterals. Second, the regenerated nodule of the cirrhotic liver is perfused, not by the portal vein, but by branches of the hepatic artery. Therefore, sudden diversion of portal venous blood through a surgical shunt does not suddenly deprive regenerated liver of its principal blood supply.

The main effects of the portacaval anastomosis are, to my mind, beneficial. In this field there have been no universally acceptable, controlled series that clearly delineate the value, or worthlessness, of venous shunts in the reduction of bleeding from esophageal varices. Because it seems that a reasonably safe and feasible operation might reduce the horrible toll of such bleeding in some patients, and because we cannot comfortably deprive patients of this hope, portacaval shunts will probably continue to be done without proper statistical validation.

### SURVIVAL

The overall mortality rate of elective shunting operations in the hands of interested and expert surgeons is now around 10 to 15 per cent. Statistically, if a group of patients with cirrhosis, in whom varices are diagnosed before bleeding begins, were observed for only two years, 80 per cent might be expected to die in that time. Although many of the deaths would not be caused directly by exsanguination, at least 10 to 15 per cent of patients having cirrhosis and varices may be expected to die eventually from their first hemorrhage.[1] About 75 per cent of patients with esophageal varices that have bled once may be expected to die within one year of the first hemorrhage, a figure which has not changed in the past 20 years. Bleeding therefore greatly lessens the predicted survival. On the other hand, of a series of patients at Massachusetts General Hospital who had a shunting procedure because of esophageal bleeding, only about one-third died within two years.[6] The mortality rate associated with splenorenal shunts was lower than with portacaval shunts, perhaps because of the greater incidence of postoperative liver failure after the latter procedure. The first year or two is the most critical period. Patients with varices known to have been present for at least two years without bleeding have a relatively good survival rate and the same is true of those who live for over two years after a shunting operation. The longer the patient survives, the less important liver disease becomes as a cause of death. In one series only about 12 per cent died of hepatic failure.

### BLEEDING AFTER SHUNT OPERATIONS

There should be no further gastrointestinal bleeding after a shunting operation. In Child's series,[5] hemorrhage recurred only when the portal venous pressure had not been adequately reduced. Nevertheless, various series show recurrent bleeding in 14 to 31.5 per cent of cases. It is generally conceded to be more common after splenorenal than after portacaval shunts. Linton[6] reported esophageal bleeding in 17 per cent of patients with splenorenal shunts and in only 3 per cent of patients with portacaval anastomosis, although only 2 per cent died of bleeding. Recurring bleeding is not necessarily a sign of portal hypertension or of closure of the anastomosis. Abdominal wall collateral veins should disappear along with any venous hums which might have been present. Esophageal varices, as visualized by barium swallow, generally disappear in patients with satisfactory decrease in portal pressure. The disappearance may take from three to six months. Their presence does not necessarily signify portal hypertension, but if the x-ray films show residual varices beyond six months after operation, the effectiveness of the operation may be questioned.

### LIVER FUNCTION

Even in persons without liver disease, laboratory tests of liver function become mildly abnormal after such minor operations as herniorrhaphy or vein stripping and somewhat more abnormal after major upper abdominal operations such as gastrectomy. If the patient has cirrhosis, this immediate deterioration in results of standard tests of liver function is more severe. The changes include a fall in serum albumin and rises in serum bilirubin, alkaline phosphatase and bromsulphalein retention. Usually within ten to fourteen days the values have reverted to preoperative levels. In patients with chronic liver disease there is apparently no greater deterioration after portacaval shunt than after operations of comparable severity in the upper abdomen. The changes in liver function tests are probably related more to the preoperative state of liver function than to the operation itself. In patients who survive the operation there is no significant worsening in results of tests of liver function over the ensuing five years. The test values, moreover, have not been helpful in predicting the well-being of the patient. Indeed, many a patient who feels vastly better after operation has distressingly abnormal results of liver function tests.

### EFFECTS OF SPLENECTOMY

The relief of portal hypertension by portacaval shunt is usually attended by decrease in size of the spleen. However, there is no consistent improvement in the granulocytopenia or thrombocytopenia that is associated with congestion of the spleen. There are isolated examples of improvement in hypersplenism, even when the spleen has been left in after a portacaval shunt. But the majority of investigators agree with MacPherson[7] that such improvment is unusual without splenectomy. MacPherson noted evidence of improved resistance to repeated respiratory tract infections in three such patients following the addition of splenectomy. The splenorenal shunt is, of course, preferred by some observers because of the splenectomy which it entails, removing both the splenic arterial inflow into the portal system and the short gastric veins which feed into esophageal varices. Splenectomy has been attended by only a 7 per cent incidence of thrombosis extending into the splenic and portal veins, an incidence no higher than that in persons with hepatic cirrhosis who are not operated on. Splenectomy does not usually alleviate the Coombs negative reduction in erythrocyte survival observed in some persons with cirrhosis of the liver.

### CHANGES IN PORTAL PRESSURE

Usually when thrombosis closes the lumen at the site of anastomosis, the event occurs in the immediate postoperative period. The complication is commoner after splenorenal anastomosis and side-to-side portacaval shunt. If this occurs, or if the shunt has not been effective in reducing the portal venous pressure below 30 cm. of water or so, the operation may be considered a failure, whatever happens to the patient. But usually this pressure is not accurately known. It may be determined, if desired, by recording the splenic pulp pressure percutaneously in patients who have had portacaval shunt or the wedged hepatic venous pressure in cirrhotic patients who have had splenectomy.

Before a surgical shunt is created, the portal venous pressure is highest in persons in whom the greatest number of spontaneously induced collaterals have formed. Therefore the latter are not effective ordinarily in decompressing the portal hypertension. Furthermore, the portal venous pressure is higher in patients with varices that bleed than in those with varices that have not bled. So it seems desirable to reduce portal venous pressure. Following portacaval shunt, Rousselot[11] obtained an average fall of 22 cm. of water to a final pressure of 22 cm. The decrease after splenorenal anastomosis was somewhat less, the average reduction having been 15 cm. of water to a final pressure of 24 cm. Reynolds[10] observed a fall in both the portal venous pressure and in the wedged hepatic venous pressure. Sherlock[12] reported a decrease in the splenic pulp pressure. But the pressures were not reduced to normal, and therefore the hepatic vascular resistance at the postsinusoidal level was maintained.

### FALL IN LIVER BLOOD FLOW

There is also a fall in the estimated hepatic blood flow after venous shunt operations. After splenorenal shunt, this decrease is of the order of 25 per cent, whereas after portacaval anastomosis it is about 40 per cent.[10,12] This amounts to a reduction of around 700 ml. per minute in flow of blood after portacaval shunts. While this represents a respectable decrease in blood flow, it must be recognized that after the operation the hepatic artery is carrying a greatly increased proportion of the total blood flow, especially to the regenerated liver tissue. Despite the decrease in hepatic blood flow, there is no measurable fall in oxygen consumption by the liver.[4] This is accomplished by a greater extraction of oxygen from a given volume of blood. The hepatic venous oxygen content after shunts, for example, is usually less than 10 volumes per cent. One might presume from such information that the parenchyma supplied by the distal end of the hepatic circulation might suffer from hypoxia, but such a presumption is belied by the fact that bromsulphalein extraction is well maintained and usually there is no progressive worsening in liver function as a result of the shunt.

### FLUID RETENTION

If the patient had had ascites before operation, it tends to recur briefly in the immediate postoperative period. Edema at the ankle on dependence of the extremity sometimes develops or increases after portacaval shunt. This is not necessarily accompanied by an increase in the pressure in the inferior vena cava, and it is seldom much of a clinical problem. These evidences of fluid retention are curious, for an increasing number of patients are being subjected to portacaval shunts for the relief of ascites. In cases in which the operation for this purpose succeeds, a change in the ratio of urinary sodium to potassium occurs consistent with relief of a hyperaldosterone effect—that is, more sodium and less potassium is excreted.[9]

### BEHAVIOR AND STUPOR

A problem in the behavior of a neuropsychiatric nature occurs in some 15 to 20 per cent of patients after portacaval shunt.[13] Von Eck described "meat intoxication" in dogs late in the last century, and the problem and even the mechanism have been observed in men for hundreds of years. In 1602

Shakespeare alluded to this in *Twelfth Night:* Sir Toby Belch says to his friend, Sir Andrew Aguecheek: "Oh, Knight! Thou lackest a cup of canary; when did I see thee so put down?" And Sir Andrew answers: "Never in your life, I think; unless you see canary put me down. Methinks sometimes I have no more wit than a Christian or an ordinary man has; but I am a great eater of beef, and I believe that does harm to my wit."

There is no sure way of predicting the incidence of such encephalopathic states. Surely a patient who has had it preoperatively may be more liable to it after a shunt is performed. Unfortunately the various predictive tests applied have given uncertain correlation. These include symptoms from ingestion of methionine, a prolonged elevation of venous blood ammonia after oral ammonium administration, and intolerance to diets containing 120 gm. or more of protein per day. Apparently behavior problems of this order follow portacaval shunt far more commonly than they do splenorenal shunt.

The usual onset of these symptoms is several weeks to months following the creation of the shunt. At first the patients may not notice that they are drowsier and occasionally confused. There is a simple disorganization of thinking with poor retentiveness and poor cooperation with complex requests. The family may appreciate this earlier than the patient, but the patient generally realizes that something is amiss. Later there is a slowing of physical movements and the development of a non-intention, flapping tremor when activating the muscles of the outstretched, extended fingers, lips or head. This flapping tremor is not specific to chronic liver disease, for it also occurs in uremia and respiratory alkalosis. The tremor, interestingly, is not seen in patients or animals with an Eck fistula. As the condition progresses, there may be twitching or even convulsions with variable deep tendon reflexes, inconstant extensor plantar responses, hypertonicity of the muscles, rousable stupor, the specific fetor and, finally, deep coma. Unless other central nervous system disease is present there are no persistent lateralizing neurological signs.

The subtle, early changes may be unmasked by certain discriminative tests. The patient may be asked to write an unfamiliar phrase each day in his record, which may divulge deterioration in his handwriting. With giggling futility, he may find himself completely unable to copy the examiner's example of a five-pointed star constructed from wooden matches. Unless he has long been familiar with mathematics, he is likely to stumble badly at subtracting seven serially from 100. The electroencephalogram is probably the most sensitive means of quantitating the problem. During the confused stage, 5 to 6 per second waves of about 50 microvolts appear, spreading from original sites in the frontal areas. As stupor deepens, the voltage increases and the rate of this diffuse delta activity slows to 2 to 3 per second. In deep coma, triphasic waves may appear. Such reversible patterns have also been noticed in vitamin $B_{12}$ deficiency, hypokalemia and hypoglycemia, but not in experimental Eck fistula.

Various precipitating factors have been demonstrated. Tolerance to barbiturates and opiates is notoriously poor in persons with hepatic cirrhosis. Eight milligrams of morphine can slow the electroencephalographic waves in persons with a tendency toward coma without altering the blood pH or the potassium or ammonia content, according to Sherlock.[13] Effective diuretic programs may also produce electroencephalographic changes and clinical precoma by leading to huge potassium deficits. Progressive encephalomalacia from arteriosclerosis renders the older patients more susceptible. Hypoglycemia is poorly tolerated. These factors are not influenced by protein restriction or by administration of neomycin.

Whenever excessive nitrogen reaches the increased bacterial flora of the small intestine in persons with cirrhosis, whether it is due to dietary protein or gastrointestinal bleeding, this neuropsychiatric syndrome may result. Oral administration of ammonium chloride, methionine and cation exchange resins which swap ammonium for sodium and potassium may also initiate such deterioration. Precoma sometimes follows use of amine-oxidase inhibitors, but most investigators, finding no elevation in blood amines, doubt that the latter contribute to this state.

Among 21 candidates with portacaval shunt and chronic liver disease, Sherlock[13] noted eight with episodic stupor, four with abnormal electroencephalograms, three whose EEGs were labile to oral ammonium chloride, and only six with normal tracings. Perhaps some sensitization of the brain is necessary before patients react. Such factors probably include preexisting cerebral disease, ammonia, unconjugated bilirubin, serotonin deficiency, disordered carbohydrate metabolism, anoxemia, narcotics, infection and hypokalemic alkalosis.

The treatment of porta-systemic encephalopathic states after portacaval shunt is usually at least partially successful. Dietary protein should be limited to what the patient can tolerate; 30 to 40 gm. a day ordinarily maintains a positive nitrogen balance. Precipitating factors must be eliminated. Intestinal antiseptics such as neomycin, 4 gm. daily, usually suppress offending coliform flora. Potassium, carbohydrate and vitamin $B_{12}$ deficits must be replaced. Hemodialysis, glutamic acid, arginine, and dilantin are generally worthless.

### GENERAL HEALTH

Finally, what is the capacity of these patients to carry on after operation? In the Massachusetts General Hospital series,[6] 82 survivors were observed for periods of from one to ten years. Two thirds of them were doing well, unaware of physical limitations and able to undertake full work or household duties. One fourth were reasonably active but handicapped by some physical limitation, half of the time due to progressive cirrhosis. Only 7 per cent remained incapacitated by cirrhosis. Many, despite exhaustive preoperative medical treatment, seemed to be much better postoperatively than before. These changes included an improved feeling of well-being and strength, restoration of normal skin color, return of normal fullness of the face, weight gain, restoration of normal musculature, and regrowth of previously sparse axillary and pubic hair. Venous shunt operations may lead to improvement in organ systems such as the small intestine as well as in the liver itself.

While this improvement occurs at a high price on the contemporary medical market, the drawbacks which have been outlined should not deter physicians from offering portacaval shunt as proper and judicious therapy.

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# Bronchial Carcinoma

## Lobar Distribution of Lesions in 250 Cases

L. HENRY GARLAND, M.D., San Francisco

• **In a study of the lobar distribution of tumors in 250 consecutive cases of primary bronchial cancer, it was noted that 130 of the tumors originated in the upper lobes, 11 in the right middle lobe, and 49 in the lower lobes. Some 40 arose in the main bronchi, and most of the remainder were either "hilar" or unspecified in anatomic location.**

**There was no apparent correlation of the lobar site of these tumors with the lobar location of childhood pneumonic lesions as observed in another group of patients in the same hospital.**

SOME INVESTIGATORS have suggested that antecedent pulmonary infection may be a factor in the development of primary carcinoma of the lung, alongside such other potential factors as constitutional defect, cigarette smoke, irritation from air pollutants, virus infection, age and a suitable hormonal milieu. In discussing the problem of precancerous lesions, Stewart[4] recently observed: "The so-called terminal bronchiolar carcinomas seem on occasion to develop in old cicatrices, sites of obsolete tuberculosis, or old pulmonary infarcts. Thus they are an example of scar cancers . . . rare . . . as precancerous lesions."

Since two of my colleagues in the Department of Radiology at the San Francisco General Hosital recently studied the lobar distribution of *pneumonia* in young persons in that hospital,[2] it occurred to me that it might be of interest to examine the lobar distribution of primary bronchial carcinoma in adults seen in the same institution.

### MATERIAL

Patients with microscopically verified primary carcinoma of the lung who were referred to, or seen by, members of the Radiological Service of the Hospital constitute the source of the material for this report. Commencing with the cases indexed in the department as recently as May 1960, a consecutive series was withdrawn from the departmental files until 250 cases were available. The age range was 35 to 85; the sex ratio 5 males to 1 female; about two-thirds of the patients were white; the remainder were negro or oriental.

### METHOD

The location of the bronchial carcinoma was determined by one or more of the following types of examination: Radiologic, bronchoscopic, thoracotomy and autopsy. Autopsy records were available for approximately one-third of the cases.

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Submitted June 15, 1960.

Main bronchus tumors were listed separately from lobar and segmental bronchus tumors. In a number of cases the site of origin of the tumor could not be determined. In three cases there was reported to be bilateral involvement.

### RESULTS

The right lung showed more frequent involvement than the left. A majority of the lesions occurred in the upper lobes. Most apparently arose in the lobar or segmental bronchi. (See Table 1.)

**TABLE 1.—*Lobar Distribution of Primary Bronchial Carcinomas in 250 Cases***

| | No. Patients |
|---|---|
| Right upper lobe | 67 |
| Right middle lobe | 11 |
| Right lower lobe | 30 |
| Right main bronchus | 18 |
| Right intermediate bronchus | 1 |
| Right hilum | 1 |
| Right lung, site unspecified | 5 |
| Total, right | 133 |
| Left upper lobe | 63 |
| Left lower lobe | 19 |
| Left main bronchus | 21 |
| Left hilum | 3 |
| Left lung, site unspecified | 6 |
| Total, left | 112 |
| Bilateral lesions of approximately equal size, or upper lobe lesions | 2 |
| Bilateral main bronchus lesions, involving carina and trachea | 1 |
| Site unspecified | 2 |

**TABLE 2.—*Percentage Lobar Distribution of Primary Bronchial Carcinomas***

| Site | Number of Patients | Per Cent of Series | |
|---|---|---|---|
| Right upper lobe | 67 | 27 | |
| Right middle lobe | 11 | 4 | |
| Right lower lobe | 30 | 12 | 53 |
| Right main bronchus | 18 | 7 | |
| Other (see Table 1) | 7 | 3 | |
| Left upper lobe | 63 | 25 | |
| Left lower lobe | 19 | 8 | 44 |
| Left main bronchus | 21 | 8 | |
| Other (see Table 1) | 9 | 3 | |
| Bilateral | 3 | 1 | |
| Unspecified | 2 | 1 | |
| | 250 | | |

The percentage distribution of lesions is summarized in Table 2. The ratio of upper to lower lobe involvement was approximately two and a half to one.

The histologic diagnoses in this group of cases were recorded as follows:

| | Per Cent |
|---|---|
| Squamous cell carcinoma | 33 |
| Anaplastic carcinoma | 30 |
| Adenocarcinoma | 10 |
| Alveolar carcinoma | 2 |
| Unclassified or mixed carcinoma | 25 |

## DISCUSSION

It is evident that in this small group of cases, bronchial carcinoma was seen a little more frequently in the right than in the left lung (approximately 133 to 112), and that the upper lobes showed more frequent involvement than the remaining lobes. This coincides with the experience reported by Ochsner in 1453 cases.[3] In that series the right lung was involved slightly more often than the left, and in almost 60 per cent of the cases the upper lobe was involved. Ochsner recorded the apparent bronchial site as follows: Main bronchus, approximately 10 per cent; lobar bronchus, approximately 66 per cent; peripheral bronchus, approximately 24 per cent. We are currently investigating a larger series of cases in an attempt at more precise determination of the site of origin of "lobar" tumors.

In the previously mentioned study of lobar pneumonia in young persons,[2] Heald and Coulson found that 79 per cent of the lesions occurred in the right lung and 21 per cent in the left; about 41 per cent were in the upper lobes. It is evident that there is not a good correlation between the location of antecedent lobar pneumonia in the young people in this hospital, and primary bronchial carcinoma in the independent older group.

It is also evident that a majority of the lesions were recorded as occurring in the lobar and segmental bronchi, rather than in the trachea and main bronchi (where Auerbach[1] found a significant incidence of "smoking" lesions which he personally interpreted as metaplasia and carcinoma in situ—an interpretation disputed by other pathologists). Only two of 250 cases showed carcinoma that might be considered as being multicentric, as contrasted with Auerbach's frequent finding of multiple bilateral lesions of "noninvasive carcinoma." The latter, of course, may be reversible changes, or may in fact seldom progress to clinical invasive disease.

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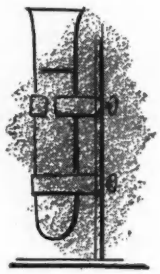

# Drug Dangers and Reactions

RALPH W. WEILERSTEIN, *M.D.*, San Francisco

• **The protection of the consumer against dangerous, adulterated, and misbranded drugs provided by the Federal Food, Drug, and Cosmetic Act has failed in some instances. A general program of reporting adverse drug reactions has been initiated on a pilot basis. Arrangements are being made to extend this program into larger hospitals. Better and more complete reporting of adverse drug reactions together with tightening of the Food and Drug law regarding new drugs will improve this situation.**

**Recently the president of the National Academy of Sciences appointed a committee at the request of the Secretary of Health, Education, and Welfare to review the policies and procedures used by the Food and Drug Administration in reaching decisions and to present recommendations. This committee has completed its work and has made specific recommendations that would give the Food and Drug Administration authority to require proof of efficacy as well as safety of all new drugs, and would provide it with sufficient resources to meet the responsibilities assigned to it.**

THE FEDERAL FOOD, Drug and Cosmetic Act is intended to protect the consumer against dangerous, adulterated and misbranded drugs and to provide for the safety of new drugs introduced into interstate commerce. In the 22 years since the passage of the Federal Food, Drug and Cosmetic Act of 1938, there have been many situations in which the protection that this act is intended to provide somehow failed.

The original reason for the inclusion of a new drug section in this law was the so-called Elixir of Sulfanilamide® incident. In that case, a manufacturer, attempting to improve his vehicle for the ingestion of sulfanilamide, used an excellent solvent—diethylene glycol—which was tasteless and odorless. Its only drawback was that it was also highly toxic. One hundred eight persons lost their lives because sulfanilamide, diluted with diethylene glycol (which is also the active ingredient in some makes of antifreeze), was introduced as a medicine without proper testing.

After the passage of the 1938 act, it was found that safety might be a goal that is not always attained. One day, the editor of a nationwide medical publication received a manuscript entitled "Coma Due to Sulfathiazole." The authors, two well known eastern physicians, were reporting for the information of the medical profession that several of their patients had gone into coma when given sulfathiazole. This was at a time when sulfathiazole was still a new drug. The editor, sensing some possible discrepancy here, referred the matter to the Food and Drug Administration. Investigation revealed that one of our country's leading drug manufacturers was allowing the "tailings" from batches of phenobarbital (of a famous brand) to slip into the sulfathiazole tablets, just as the corner butcher may let you have some of the hamburger left in the meat grinder from the previous customer. This situation was easily corrected. Another very well known manufacturer of fine chemicals and drugs happened one day to get his cadmium chloride mixed with his calcium chloride because of a labeling mixup, and still another confused cream of tartar with tartaric acid. Fortunately, these errors were detected before the articles reached the consumer.

One of the most interesting and one of the more sophisticated misbrandings grew out of discovery by one of our largest drug manufacturers that a drug, chemically known as carbaminoyl choline (a cholinesterase inhibitor), would be effective in a dosage of 0.25 mg. as a liquid in a 1 cc. ampul for the relief of postoperative distention. The ophthalmic preparations department of the same firm also found that this same drug, dispensed in 1 cc. ampuls, in powder form, in the amount of 0.15 gm., was an excellent drug for the topical treatment of glaucoma. Both drugs were put out by the same firm in 1 cc. ampuls, one a liquid, the other a powder; both were found to be invoiced as 1 cc. ampuls, and because of the confusion in identifying the proper products on the wards of hospitals, in pharmacies, at wholesalers and all along the line, 15 persons suddenly died unexpectedly. In none of these cases did the attending physician realize what the cause of death was until we made our investigation.

Then it was discovered that the fatal dose of this drug was much less than 150 mg., which was 600 times the intended injectable dose. Death was by pulmonary edema and collapse. A careful control of the production and marketing operation would have prevented these deaths. A criminal prosecution brought a plea of guilty and a maximum fine, but this did not restore life to the deceased.

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Submitted November 21, 1960.

A few years ago the phenothiazine tranquilizers achieved tremendous publicity as the answer to the problem of mental illness. We know, of course, that the performance was less than the promise, but there was wide use of them—and there still is, as adjuvant therapy, in this disease.

From one hospital, and from one medical school and then from another, we began to receive scattered reports of an "idiosyncrasy to Chlorpromazine," which turned out to be liver damage. Each physician observing such a case thought he was the only one. At one time, it was estimated that liver damage developed in from 1 per cent to 5 per cent of all patients receiving this drug. The Food and Drug Administration was able to require warnings and information concerning the hazards inherent in the use of these drugs, and this information has become common knowledge to the American physician.

The staff available to control and review all the claims, all the evidence as to safety of the drugs produced in this country is pitifully small. A handful of doctors in Washington review new drug applications; an even smaller handful look over the reports concerning existing drugs. There is just being set up a program for general reporting of adverse drug reactions so that there may be at least a central reservoir of information concerning what the drugs—old and new—are doing in the way of unusual reactions.

Five years ago, the Food and Drug Administration began a pilot study on the reporting of adverse drug reactions from ten eastern and midwestern hospitals. From this pilot study, there has grown a program which this year was expanded to bring in reports from various places around the country, where adequate records are kept, of adverse drug reactions as they occur. With the planned expansion of the reporting system, the medical profession and the Food and Drug Administration can learn promptly just what the population of this country is encountering in the way of drug reactions; as soon as these reports are received steps may be taken to protect the physician and the patient from failure of the drug labeling to bear appropriate warnings or instructions.

The Food and Drug Administration is at present attempting to set up such arrangements with several of the larger hospitals in this area. It is also looking at the laws under which it operates and is finding, with the help of interested physicians and others, that these laws may need strengthening, for they were drawn up at times and in situations vastly different from the age of modern science and technology which has changed the whole character of drug production in the past two decades.

Recently, the president of the National Academy of Sciences appointed a committee, at the request of the Secretary of Health, Education, and Welfare, to review the policies and procedures used by the Food and Drug Administration in reaching decisions and to present recommendations. This committee has just completed its work and it has made the following recommendations:

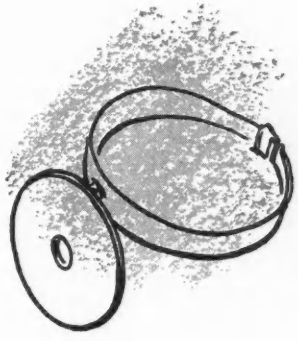
1. The FDA should be given statutory authority to require proof of the efficacy, as well as the safety, of all new drugs. Treatment of a patient with an ineffective drug in place of an effective one may jeopardize his recovery. This is true even though the drug may not be intrinsically harmful, and even though the specific condition for which the drug is given may not be ordinarily regarded as life-threatening.
2. The FDA should be given statutory authority to require manufacturers of new drugs to maintain records and submit reports of clinical experience and other relevant data, not only before but after the drug is released for sale, as requested by the Commissioner in his proposed Factory Inspection and Drug Amendments of 1960. Any evaluation of a new drug is subject to revision in the light of broader experience, and the FDA must be in a position to advise the profession and warn the public promptly whenever new hazards are revealed.
3. The FDA should be given statutory authority to apply certification procedures to all antimicrobial agents used in the prophylaxis and treatment of infectious diseases. The Committee sees no reason for limiting certification to those antibiotic preparations which happen to have come on the market prior to 1950, and further believes that all agents employed for equally serious conditions should be subject to equivalent measures of control.
4. The Committee recognizes the importance to the public health of ensuring that all drugs are prepared under the highest standards of quality control. It, therefore, endorses the Commissioner's proposals in the Factory Inspection and Drug Amendments of 1960 to clarify and strengthen existing inspection authority, and to require that all drugs be manufactured and packaged under adequate controls.
5. The Committee believes that the information supplied to physicians concerning drugs should be not only accurate, but also complete, and that the date of such information is essential to its proper evaluation. It, therefore, endorses the proposed amendments to present labeling requirements published by the Commissioner in the Federal Register for 22 July 1960.
6. The Committee considers that the advertising of pharmaceuticals requires more careful regulation than that of products unrelated to the

prevention and cure of disease. It, therefore, recommends that careful study be given to the problem of coordinating the supervision of labeling, promotional material, and other advertising of drugs, now divided among several agencies of the government, and to means of ensuring that all information concerning drugs conveyed to the profession and the public by whatever media be in conformity with scientific fact.

7. When a decision has been reached concerning an application, a statement should be prepared and incorporated in the file summarizing the conclusions, the names and opinions of those involved, and such other data as may be necessary to provide a concise record of the basis for the decision. This should be of great assistance in the administrative review of current actions and in the scientific review of the files in relation to subsequent applications, as well as when the advice of consultants is sought.
8. The staff members responsible for processing applications should be supported to the utmost in their efforts to obtain submission of truly dependable scientific information on the efficacy and safety of the products. The data initially submitted by the manufacturer are not always of sufficient quality and quantity to permit a sound decision as to the merits of the product.
9. The FDA should be strongly supported in its effort to maintain a research program of high quality on the methodology and standardization of drug testing and related areas of basic science. This is important not only to improve the methods available for carrying out its responsibilities to the public but also as an aid in recruiting and retaining competent scientists on the staff.
10. The Committee urges the Commissioner to seek such authorization as may be necessary to establish an advisory organization of scientific and technical experts as a recognized resource for advice on criteria, procedures and policies for the execution of the responsibilities of the FDA.
11. It is recognized that these various recommendations cannot be carried out without expanded resources, both of funds and of personnel. The committee also considers that the present resources of the FDA are less than adequate to meet existing responsibilities. It, therefore, urges that the FDA be granted the authority and funds required to employ and retain larger numbers of highly qualified personnel and to support their activities, and endorses the recommendations made to this end by the Citizens' Advisory Committee in 1955.

The Secretary of the Department of Health, Education and Welfare has concurred with all these recommendations, except No. 6, which he has requested be given further study. When these recommendations are implemented, there will be, I hope, fewer drug reactions and dangers, and those that do occur will be recognized much sooner.

Department of Health, Education, and Welfare, Federal Office Building, Fulton and Leavenworth Streets, San Francisco 2.



# Dangerous and Hypnotic Drug Act

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IN 1945, California enacted the Dangerous Drug Law. Said to be the most comprehensive regulatory measure concerning control of hypnotic drugs,[1] it is a part of the Pharmacy Law and is administered by the State Board of Pharmacy.

Section 4211 of the Business & Professions Code defines *Dangerous and Hypnotic Drugs:*

" 'Dangerous Drug': 'Hypnotic Drug.' 'Dangerous drug' means any drug unsafe for self-medication, except preparations of drugs defined in subdivisions (e), (f), (h), and (i) hereof, designed for the purpose of feeding or treating animals (other than man) or poultry, and so labeled, and includes the following:

"(a) Any hypnotic drug. 'Hypnotic drug' includes acetyluria derivatives, barbituric acid derivatives, chloral, paraldehyde, sulfonmethane derivatives, or any compounds or mixtures or preparations that may be used for producing hypnotic effects.

"(b) Aminopyrine, or compounds or mixtures thereof.

"(c) Amphetamine, desoxyephedrine, or compounds or mixtures thereof except preparations for use in the nose and unfit for internal use.

"(d) Cinchophen, neocinchophen, or compounds or mixtures thereof,

"(e) Diethyl-stilbestrol, or compounds or mixtures thereof.

"(f) Ergot, cotton root, or their contained or derived active compounds or mixtures thereof.

"(g) Oils of croton, rue, savin or tansy or their contained or derived compounds or mixtures thereof.

"(h) Sulfanilamide or substituted sulfanilamides, or compounds or mixtures thereof, except preparations for topical application only containing not more than 5 per cent (5%) strength.

"(i) Thyroid and its contained or derived active compounds or mixtures thereof.

"(j) Phenylhydantoin derivatives.

"(k) Any drug which bears the legend: 'Caution: federal law prohibits dispensing without prescription.'

"(l) Hypnotic drugs when combined and compounded with non-hypnotic drugs."

[1] *The Narcotic Problem*, edited by W. L. Prosser, 1 U.C.L.A. Law Review 405-546.

**• It is unprofessional conduct within the meaning of the Medical Practice Act to prescribe a dangerous drug without either a medical examination by a physician or other medical indications.**

**Dangerous and hypnotic drugs are specifically defined by both state and federal law and distribution is strictly regulated.**

**A physician may administer to his own patients such amounts of dangerous drugs as are necessary for the immediate needs of the patient. The physician may obtain such needed amounts of these drugs by an order placed with a pharmacist marked "for administration to immediate needs of patients." A licensed physician may also prescribe dangerous and hypnotic drugs for patients and such prescriptions may be refilled on the specific authorization of the physician.**

**A physician who dispenses dangerous and hypnotic drugs to patients must obtain a hypnotic drug license; he must use specific purchase orders when purchasing; the drugs must be labeled in the manner provided by law; and all records of sale shall be open to inspection by authorized officers of the law and kept for three years.**

**By the Principles of Medical Ethics physicians are bound to limit the source of their professional income to medical services actually rendered. Recent decisions of the Judicial Council answer questions concerning a physician's interest in a corporation which purchases, packages and sells medicines under a corporate name. Also answered are questions as to the measures that exist to prevent physicians from abusing the privilege of owning a pharmacy, and whether a physician can ethically rent space in a building owned by him to a pharmacist with a percentage of the income of the pharmacy as rental.**

**The public welfare of California and the nation as determined by legislation strictly regulates the distribution of dangerous drugs. It is in the best interest of the medical profession and its patients that these laws be understood and carefully observed.**

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The Federal Food, Drug and Cosmetic Act (FFDC) defines and regulates narcotic and hypnotic drugs by prohibiting, among other things, the *misbranding* of them. Section 502(d) provides that a drug shall be deemed to be misbranded—

"If it is for use by man and contains any quantity of the narcotic or hypnotic substance alpha-eucaine, barbituric acid, beta-eucaine, bromal, cannabis, carbromal, chloral, coca, cocaine, codeine, heroin, marihuana, morphine, opium, paraldehyde,

peyote, or sulfonmethane; or any chemical derivatives of such substance, which derivative has been by the Secretary, after investigation, found to be, and by regulations designated as, habit forming; unless its label bears the name, and quantity or proportion of such substance or derivative and in juxtaposition therewith the statement 'Warning—May be habit forming.'"

The problem of addiction caused by the use of narcotics has been recognized for centuries. Sedatives or hypnotics are drugs recently developed. Chloral hydrate is said to be the oldest. It was discovered in 1832 and introduced into medicine in 1869 by Liebreck. A German scientist, in 1907, discovered derivatives of barbituric acid while working with coal tar. Winthrop & Co. brought the drug Veronal, a barbiturate, to this country in 1907. It was not until after World War I that any large production of barbiturates was begun in this country.

The misuse of them has presented serious problems in recent years. Among habitual users, these drugs are called: *red birds, red devils, yellow jackets, brownies, green dragons, blue bombers, violets* and *goofballs.*

There is a lively dispute among authorities as to whether the hypnotic drugs are properly classified as addictive. In public policy of the State of California, hypnotic drugs are classified as dangerous and their distribution is strictly regulated.

It is well established that a state, in the exercise of its police power, may regulate and control the manufacture, sale, distribution and possession of drugs in the interest of public health and welfare.[2]

Section 4051 of California's Business & Professions Code provides that this "chapter" of the law which concerns licensing of pharmacists and manufacturers of drugs "does not apply to or interfere with anyone who holds a physician's and surgeon's certificate or certificate to practice chiropody and who is duly registered as such by the Board of Medical Examiners or the Board of Osteopathic Examiners of this State, with supplying his own patients with such remedies as are necessary in the treatment of the condition for which he attends such patient if he acts as their physician and is employed by them as such and provided such person keeps accurate records of drugs dispensed and that such drugs may not be dispensed by a nurse or attendant."

The Dangerous and Hypnotic Drug Law, by Section 4226, provides: "This article does not require a license from and shall not apply to or interfere with a physician, dentist, chiropodist or veterinarian in administering hypnotic drugs to his own patients. Such hypnotic drugs shall be administered only by the physician, dentist, chiropodist or veterinarian."

[2]Minn. ex rel Whipple v. Martinson 256 U. S. 41 (1920). In re Yun Quong 159 Cal. 508, 114 Pac. 835 (1911).

Section 4226 is an exception to Section 4222 which provides that "every person who furnishes any hypnotic drug to any other person shall first obtain from the board [State Board of Pharmacy] an hypnotic license."

Section 4213 defines the word *administer* as used in the article to mean "the furnishing by a physician and surgeon, dentist, chiropodist or veterinarian *to his patient* of such amount of drugs or medicines referred to in this article *as are necessary for the immediate needs of the patient.*" "Administering" and "furnishing" drugs for the "immediate needs" of the patient would appear to be different from "dispensing" or prescribing for the general need of the patient. An exact definition of a patient's "immediate needs" is not provided by the law. But each physician, where furnishing dangerous drugs for the immediate needs of the patient, ought to use some reasonable rule of thumb such as twelve hours or twenty-four hours or some minimal dosage as his standard of judgment of what constitutes sufficient dangerous drugs for the immediate needs only of his patient.

After Section 4226 was enacted, the State Board of Pharmacy asked the Attorney General[3] whether a practitioner "who administers hypnotic drugs to a patient will be required to obtain a hypnotic drug license and purchase hypnotic drugs upon hypnotic drug purchase order form."

In answer to this question, the Attorney General considered three different situations:

### A. *Administration of Hypnotic Drugs to His Own Patients*

When a physician administers, to his own patient, such amounts of hypnotic drugs as are necessary for the *immediate needs* of the patient, he is not required to have a hypnotic drug license or required to purchase those hypnotic drugs upon a hypnotic drug purchase order form.

Rule 1745 of the State Board of Pharmacy provides that a pharmacy may furnish to a physician and those others named in the law, hypnotic drugs for *"administration to patients"* upon an order showing the date, address, license classification, name of the practitioner and the name, type, strength and quantity of hypnotic drugs ordered. Such order should bear the words: "For Administration to Immediate Needs of Patients" or words of like intent. Such orders are placed by the pharmacist in a special file.

The Attorney General further ruled that there is no requirement that a physician keep a separate record of the hypnotic drugs purchased or supplied to his patients which are necessary for their immediate needs. Good medical practice would dictate,

[3]18 Opinions Attorney General Cal. 209.

however, that the physician record in the patient's clinical record the hypnotic drugs administered.

It should be emphasized that hypnotic drugs shall be administered only by the physician, dentist, etc. There is only one exception to this: In any licensed or county hospital, a registered nurse, on order and direction of a physician, may administer hypnotic drugs to a patient registered in the hospital or to a patient under emergency treatment in the hospital, in accordance with Section 4225. In a hospital, a special record must be kept of the administration of hypnotic drugs, showing the amount given, the type, date given and the name and address of the person to whom administered. The law does not specifically require it, but sound administration would dictate that the name of the physician who ordered the drug and the registered nurse who administered it also be recorded. This section authorizes a hospital that does not employ a pharmacist to purchase a supply of hypnotic drugs on a hypnotic drug purchase order form in the name of the hospital.

Authority granted in this section is to "licensed or county hospitals not employing pharmacists." Section 4047 excludes from the term "licensed hospital," nursing homes, maternity homes, rest homes, sanatoriums or lying-in asylums. Such establishments would not appear to have authority to order and have on hand a supply of hypnotic drugs, unless they employed a pharmacist. It is understood that some of these facilities make a contract with a local pharmacist in order to provide a supply of needed hypnotic drugs.

B. *Dispensing or "Purchase Order Practitioner"*

Another means of furnishing hypnotic drugs is outlined in Section 4222: "Every person who furnishes any hypnotic drug to any person shall first obtain from the board [State Board of Pharmacy] an hypnotic license, etc." The board, under Section 4223, shall supply at cost a book of serially numbered triplicate hypnotic drug purchase order forms to physicians and others licensed under 4222.

The physician adopting this procedure is usually referred to as a "purchase order practitioner." He is not limited in the amount of hypnotic drugs he may purchase on an order form, nor is he restricted to furnishing an amount sufficient only for the immediate need or use of his patient.

Due to the dangerous character of hypnotic drugs, a physician should only furnish such amounts as are reasonably necessary for the condition for which he attends his patient.

Section 4228 provides that no person shall dispense any dangerous drug upon prescription except in a container correctly labeled with the date, name and address of furnisher and person for whom prescribed, and directions for use. The section specifically applies to dangerous drugs dispensed "upon prescription." Does it apply only to dispensing by a druggist upon prescription, or does it apply also to dispensing by a physician upon his own unwritten prescription? The advice to a physician on this point is: Follow the provisions of this act. Because of the wording of the statute, you might not technically be convicted of violating the criminal provisions of the act if you did not properly label dangerous drugs furnished, but good public policy and sound medical practice dictate that you should observe the precautions of the law concerning labeling dangerous drugs you dispense.

Section 4224 provides the method by which a physician who dispenses hypnotic drugs shall order them. The purchase order forms furnished by the State Board of Pharmacy to licensees are filled out in triplicate showing the date, name of supplier, name and quantity of hypnotics ordered and signature, license number and address of licensee. The original and one copy of this form are sent to the supplier, who must forward the duplicate copy to the State Board of Pharmacy within thirty days, and the third copy is kept by the physician for three years. If the drugs are purchased out of state, then the original is sent to the supplier and the duplicate is sent directly to the State Board of Pharmacy.

Recently, the attorney general of an eastern state uncovered an extensive counterfeit drug operation which was nationwide in scope. Analysis showed that well-known brand names were affixed to products that were not up to standard and were harmful in some instances. The counterfeiting was so skillful that some drugs could not be recognized as bogus except by chemical analysis. These drugs were "bargains" sold at less than regular prices by an alleged jobber.

Physicians who are *furnishing* or *dispensing* by sale hypnotic drugs in quantities greater than for the immediate needs of patients, are assuming certain legal and practical responsibilities which must be well understood.

C. *Furnishing by Prescription*

The third method by which a physician may furnish dangerous hypnotic drugs to his patients is by prescription, after examination of the patient or upon medical indication therefor. It is unprofessional conduct within the meaning of the Medical Practice Act to prescribe a dangerous drug without either a medical examination by the physician or other medical indication. This would be construed by the courts to mean that ordinarily a medical examination of a patient must be made before a dangerous drug is prescribed. However, other medical indications also give him authority to prescribe dangerous drugs.

If a patient is in the hospital, a nurse or intern might report facts of an emergency nature upon which a physician could reasonably prescribe immediately needed dangerous drugs. Physicians associated together are frequently familiar with the medical problems of each other's patients, even though each might not have examined all of the other's patients. In emergencies and when they serve for one another on days off or at times of sickness or vacation, one of them might be called upon to prescribe for an associate's patient whom he had not examined. Yet from information available to him through the office records and consultation, he might have sufficient medical indication to prescribe a dangerous drug in an emergency. These examples are given as guides as to what is meant by the words "medical indication" and not as a limitation on the meaning of them.

The impression might be gained from recent articles appearing in print that a physician could comply with the law that he must examine the patient or that there must be a medical indication for the use of drugs prescribed merely by asking if the patient is nervous or by asking him to stick his tongue out.

However, the Board of Medical Examiners in applying the above requirement of the Medical Practice Act has administered it strictly and realistically. Any physician guilty of subterfuge to cover up the peddling of narcotics invariably has the book thrown at him, and in suspicious circumstances the mere showing of an entry on the physician's record that a patient needed narcotics is viewed with skepticism.

It has often been contended before the Board that a "physician's glance" could tell that a man was sick and needed narcotics. This contention has never impressed the physicians on the Board. The record of the Board indicates that it has not been fooled by phony, inadequate physical examinations or trifling medical indications. It can be expected that the Board would hear expert testimony from other physicians on the medical question: Were the drugs prescribed shown to be necessary by either a medical examination or other medical indications?

Drugs may not be dispensed by a nurse or attendant and may only be prescribed by a physician, dentist, chiropodist or veterinarian, according to sections 4051 and 4227.

Section 503(b) of the FFDC Act exempts from its labeling and packaging provisions a drug intended for use by man, such as those described in Section 502(d), when:

1. Such drug is administered by or under the supervision of a licensed practitioner; or

2. Such drug is dispensed only upon a written prescription of a practitioner licensed by law to administer such drugs.

This section provides specifically: "The act of dispensing a drug contrary to the provisions of this paragraph shall be deemed to be an act which results in the drug being misbranded while held for sale."

In a recent case, the court has applied the provisions of Section 503 to the act of dispensing drugs by a licensed physician when such drugs were not properly labeled or packaged.[4] In this case, Federal agents had purchased barbiturates from Doctor Brown. He was arrested. At his trial the jury found him guilty of dispensing misbranded drugs contrary to the provisions of Section 503. The case was appealed and the appellate court affirmed the decision of the trial court. Part of the opinion follows:

"There seems to be some concept among members of the medical profession that to have a license to deal in medicine carries a license to deal in barbiturates. That is not the law. The medical profession might just as well understand it. If I come to you for treatment and you in your medical capacity examine me, and after examination determine that certain barbiturates would be beneficial to me, then you have a right to write a prescription to me and then I have a right to get them and use them but not otherwise."

#### REFILLING PRESCRIPTIONS

Section 4229 provides that no prescription for any dangerous drug may be refilled except upon authorization of the prescriber. The authorization may be given orally, or it may be given at the time of the original prescription.

A prescription refill can only be authorized by the prescriber, such as a physician, either orally or in writing. A nurse or attendant is not authorized to order a prescription refill, but may relay an authorization given by the physician.

#### STOCKS OPEN FOR INSPECTION

Section 4231 provides that all stocks of any dangerous drug of a physician and the others mentioned in the Act "shall be at all times during business hours open to inspection by authorized officers of the law." Sections 4232 provides that all records of sale or disposition of dangerous drugs shall be open to inspection and be preserved for three years. Ordinarily, such records will not be examined unless there is an indication that the Dangerous Drug Act is being intentionally violated or disregarded.

#### VIOLATION PENALTY

Violation of any provisions of the Dangerous Drug Act is a misdemeanor punishable by fine or imprisonment. Upon conviction of a physician, he would be subject to having his license to practice revoked or suspended by the State Board of Medical Examiners.

[4]Brown v. U. S. [Texas, 1958] 250 F 2d 745.

As a matter of information, under Section 4163 of this law, a report must be made to the State Board of Pharmacy when chemical examination of tissue or an organ or body fluid shows the presence of poison. Details concerning the poison must also be reported.

For the fiscal year 1958-59, the coroners' offices of the State reported 666 cases distributed as follows:

| | No. Cases | Per Cent of Total |
|---|---|---|
| Hypnotic drugs | 428 | 64 |
| Tranquillizing drugs | 19 | 3 |
| Industrial, agricultural and household | 106 | 16 |
| Narcotics | 41 | 6.2 |
| Carbon monoxide | 48 | 7.2 |
| Miscellaneous drugs | 24 | 3.6 |

Many of these were cases of intentional suicide or attempts at self-destruction. Perhaps if drugs had not been used, some other means would have been employed. Data on cases of this kind are often used to illustrate to the public the dangerous nature of and the need for strict control of hypnotic drugs.

It is doubtful that such a conclusion could be supported in fact or logic. For example, the suicide rate in San Francisco is almost double that of Los Angeles, a difference that hardly can be ascribed to the availability of drugs.

### PRINCIPLES OF MEDICAL ETHICS

The American Medical Association's Principles of Medical Ethics provide: "In the practice of medicine a physician should limit the source of his professional income to medical services actually rendered by him, or under his supervision, to his patients. . . . Drugs, remedies or appliances may be dispensed or supplied by the physician provided it is in the best interests of the patient."

Recently, several questions concerning ethical problems relating to a physician's interest in a drug corporation, proper measures to prevent abuse of the privilege of owning such an interest, and rentals by a physician of store space to a pharmacy with a rental based on percentage of income, were answered by the Judicial Council.*

QUESTION: Is it ethically permissible for a physician to have an interest in a corporation which purchases, packages, and sells medicines under a corporate name? Is it permissible for a group of physicians to form a corporation for such a purpose?

ANSWER: It is not *in itself* unethical for a physician or a group of physicians to own or invest in the stock of a pharmaceutical company. The *Principles of Medical Ethics*, as recently as June, 1954, contained the statement "an ethical physician does not engage in barter or trade in the appliances, devices or remedies prescribed for patients, but limits the sources of his professional income to professional services rendered the patient."

*The Judicial Council has authorized the publication of the opinions.—Edwin J. Holman, Secretary.

In June, 1954, the *Principles of Medical Ethics* was revised to provide in this regard: "It is unethical for a physician to participate in the ownership of a drugstore in his medical practice area unless adequate drugstore facilities are otherwise unavailable . . ."

In June, 1955, the *Principles of Medical Ethics* was revised again to provide that "it is *not* unethical for a physician to prescribe or supply drugs, remedies, or appliances as long as there is no exploitation of the patient."

At the June, 1957, Annual Session of the American Medical Association the present *Principles of Medical Ethics* was adopted. Section 7 of the *Principles* provides that "drugs, remedies or appliances may be dispensed or supplied by the physician provided it is in the best interest of the patient."

In opinions published in *The Journal* (March 30, 1957, and Nov. 15, 1958) the Judicial Council, interpreting this principle of medical ethics, expressed the opinion that the physician as a citizen has the right to make investments according to his own best judgment and that the fact that he is a physician should not preclude him from investing in the stock of a pharmaceutical company, provided, of course, that no subterfuge is employed and no unusual control of the company is exercised by the doctor. The Council also expressed the opinion that it is not, in itself, unethical for physicians to own pharmacies, provided there is no exploitation of the patient.

QUESTION: What measures exist to prevent doctors from abusing the privilege of owning a pharmaceutical company or an interest in it?

ANSWER: In its opinions published in *The Journal* (March 30, 1957, and Nov. 15, 1958) and in its report to the House of Delegates at the Clinical (Dallas) Meeting in 1959, the members of the Judicial Council agreed that there are many variations of this practice. There are, therefore, many questions of fact to be resolved locally before a particular practice can be evaluated properly.

If a complaint is made regarding the practices of a particular physician or a group of physicians who own a pharmaceutical company, the local medical society has a positive obligation. The society must ascertain *whether the practice exploits the patient, whether it results in inferior medical care, whether the costs of drugs to the patient are increased, whether the situation is an act in derogation of the dignity and honor of the medical profession and its traditions, whether the particular practice is a subterfuge to permit the doctor to accept a rebate, whether it tends to cause the doctor to overprescribe or prescribe less effective drugs, or whether the practice permits the doctor to exercise control over the company so as to work against the best interests of the patient.*

The members of the Judicial Council agree that any complaint against a physician or group of physicians who own a small pharmaceutical company should be directed to the local medical society for investigation of the facts and for initial disposition of the ethical question involved. *Neither intentional nor inadvertent abuse of the privilege should be permitted by the medical profession.*

QUESTION: May a doctor who is owner of a building rent space in that building as a pharmacy, with a percentage of the income of the pharmacy as rental?

ANSWER: No. The rental should be a fixed one. Were the rental to be based on the amount of business, it might well be argued, and, indeed, be the case, that fee splitting existed. In addition, the temptation would be ever present for the doctor-owner to encourage patients to take their prescriptions to that pharmacy. The evils inherent in such practice are too obvious to be mentioned.

# Small Bowel Rupture from Abdominal Blows

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• **Rupture of the small bowel with nonpenetrating abdominal trauma occurs in one-third to one-fourth of all abdominal injuries. Perforation of the bowel can result from injuries of crushing, bursting or tearing type. The symptoms are those associated with peritoneal irritation. Repeated observation and examination is necessary for diagnosis. Operative repair is mandatory. Two cases, one in a child, are reported.**

RUPTURE of almost every organ within the abdominal cavity has occurred without external signs of injury. Injuries to the kidneys, spleen, liver and pancreas are the most common, these being solid, fixed abdominal viscera. From one-quarter to one-third of all abdominal injuries involve the small bowel alone.[2,8,11] Cottrell[1] noted that the intestine was injured in 10 per cent of 800 cases he reviewed. Trauma is produced by blows, falls, blasts, car collisions and similar accidents. Automobile collision is by far the most common cause of nonpenetrative abdominal trauma, impact with the steering wheel (which is also an etiological factor in liver trauma)[9] being noted frequently. The fragility of the small bowel and the fact that it can be ruptured by trauma that does not injure the skin have been recognized since the time of Aristotle. It was a common cause of death in the Middle Ages, often from kicks of horses or blows.[7]

Small bowel rupture occurs most commonly where the bowel is either firmly fixed to the posterior abdominal wall or has a short mesentery—these sites being the duodenum, the jejunum near and immediately distal to the ligament of Treitz, and the ileum just proximal to the ileocecal junction. The side most often involved is the antemesenteric border. Multiple lacerations of the small bowel occur in 10 per cent of cases.[1,3,5,12]

### MECHANISM OF THE RUPTURE

Geohegan[3] expressed belief that the majority of ruptures of the small bowel from nonpenetrating trauma are bursting injuries caused when a sudden force is applied to the abdominal wall and the increase in the intra-abdominal and intraperitoneal pressure is transmitted to the hollow viscera. He postulated that the length and tortuosity of the intestinal tube is such that the sudden increase in pressure in the mid portion may not be dissipated by release through its open ends and the pressure, reaching a maximum at a point near a turn or kink which provides a trap for the fluid and gas, causes rupture there. Poer and Woliver[10] said that most frequently such perforations result from a crushing injury in which the external force compresses the bowel against the spine or pelvic bones, but that sometimes tearing injuries may result from a violent force being applied at a tangent to the body.

### SYMPTOMS

Perforation of a hollow viscus must be suspected when there is persistent abdominal pain, spasm and tenderness, either with or without nausea or vomiting, after severe trauma to the abdomen. Signs of peritoneal irritation, with local or general abdominal tenderness and rigidity, are present. The higher the perforation, the more likely that chemical irritation is the cause of peritonitis; the lower the perforation, the more likely that peritonitis is bacterial.[6] Peristalsis is either absent or greatly diminished. Pain is severe, continuous and generalized. Pain referred to the shoulder or neck is indicative of diaphragmatic irritation due to either free air or escaped fluids. Pain referred to the rectum and perineum with associated tenesmus is frequently a sign of high rectal or sigmoid injury. Pain may be referred to the right testis as a result of irritation of the genitofemoral nerve by spillage from retroperitoneal rupture of the duodenum. A very significant character of peritoneal pain due to spilling of either hollow visceral contents or blood is the increase that occurs when the patient moves. Hemorrhage is not an outstanding feature.

Complete rupture may be single or multiple and may be associated with mesenteric contusions, tears or detachments; in such instances there will also be severe hemorrhage. Localized signs are commonly absent for three to twelve hours.

The pulse rate increases as soiling of the peritoneum progresses. It is also increased in hemorrhage and shock and is therefore significant of serious intra-abdominal injury.

From the Department of Surgery, Culver City Hospital, Culver City.
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Except in infants, gas is not normally roentgenographically visualized in the small intestine. Therefore, in perforations of the healthy small intestine, evidence of free intraperitoneal gas is the exception rather than the rule; seldom is there evidence of intraperitoneal air in cases of jejunal and ileal perforation. An exception is retroperitoneal duodenal rupture, in which retroperitoneal air may be seen to outline the psoas muscle shadow. However, free gas may be demonstrated in approximately 80 per cent of patients with perforation of the stomach or the first portion of the duodenum, and in almost all of those with perforation of the colon.[4]

The presence of free blood in the stomach indicates upper abdominal injury. If fresh or darkened blood is seen on rectal examination, intestinal injury is likely.

The leukocyte count becomes rapidly elevated in most instances of intraperitoneal hemorrhage and also in cases of chemical peritonitis from upper gastrointestinal spillage. The erythrocyte count is not dependable as an index of loss of blood. Hemoglobin and hematocrit determinations, as single determinations, are misleading.

All patients with a history of abdominal trauma should be closely observed up to 48 hours, with repeated examinations of the abdomen, pulse, blood pressure, respiration and hemoglobin and leukocyte count. There may be a high coincidence of other severe injuries such as multiple fractures and craniocerebral trauma, with the patient perhaps comatose, or complications such as alcoholic intoxication. Such complications greatly increase the difficulty of evaluating an abdominal lesion requiring immediate operation.

## TREATMENT

Surgical treatment is mandatory as soon as the diagnosis is made. The mortality rate increases with lengthening of time between rupture of the bowel and operation. The rate for jejunal perforations is twice that for ileal perforations.

Intestinal decompression and administration of antibiotics should be carried out, together with indicated blood and fluid replacement in preparation of the patient for operation. Spinal anesthesia was the most successful anesthesia in a series reported by Geohegan.[3] At operation, thorough exploration of the abdomen must be carried out even after one perforation is located, for often there are multiple perforations and other intra-abdominal injuries. The perforation in the bowel can be treated by either simple closure or by resection, depending on the extent of the involvement, with anastomosis if necessary. All contaminating material should be washed from the abdomen.

## REPORTS OF CASES

CASE 1. A 5-year-old boy ran into a parked automobile while running down a hill. This was at about 3:00 p.m. He went home complaining of severe pain in the abdomen. He vomited blood and soon afterward was taken to the office of a physician. The abdomen was rigid but not board-like. There was tenderness across the upper portion. Resonance to percussion over the liver was noted. The patient was admitted to the hospital at 5:30 p.m.

X-ray films of the abdomen showed free gas beneath the domes of the diaphragm. Leukocytes numbered 17,000 per cu. mm.—64 per cent segmented forms, 18 per cent stabs and 18 per cent lymphocytes. The hemoglobin content was 14.4 gms. per 100 cc. Erythrocytes numbered 4.53 million per cu. mm. The body temperature on admission was 98.8° F. and the pulse rate was 100 per minute.

At laparotomy a perforation 1.5 cm. in diameter was found in the jejunum 20 to 25 cm. distal to the ligament of Treitz. The ruptured area was punched out and rounded and allowed free exit of the intestinal contents into the peritoneal cavity. There was a large amount of plastic exudate over the surface of all loops of small bowel. The perforation was repaired, the surrounding area was washed with copious amounts of saline solution and the abdomen was closed. The body temperature was elevated for about a week after operation, then subsided. The patient did well thereafter.

CASE 2. A 49-year-old man was struck across the lower abdomen by a piece of 2 x 4 wood that was flipped from the rapidly whirling blade of a power saw. The time of the accident was about 9 a.m. The patient continued to work that morning but went home in the afternoon. There he vomited one to two quarts of black liquid. He had severe aching across the lower abdomen and great aggravation of abdominal pain when he moved. Soon after examination in the office he was admitted to the hospital, at 7:42 p.m. The pulse rate was 120 per minute, and the blood pressure 140/80 mm. of mercury. Board-like rigidity and pronounced tenderness of the entire abdomen were noted. There was decreased dullness to percussion over the liver, bilateral shifting flank dullness and questionable evidence of fluid wave. On auscultation bowel sounds were absent.

X-ray films of the abdomen showed free gas under the domes of the diaphragm.

Leukocytes numbered 11,900 per cu. mm.—80 per cent segmented forms, 15 per cent stabs and 5 per cent lymphocytes. The hemoglobin content was 16.7 gm. per 100 cc. Erythrocytes numbered 5.4 million per cu. mm. The rectal temperature was 100.2° F. Results of urinalysis were within normal limits except for the presence of acetone.

A Miller-Abbott tube was inserted and the patient was taken to surgery. At laparotomy there was a plastic exudate over all loops of the small intestine and a large collection of brownish-green fluid in the pelvis. A perforation 0.5 cm. in diameter with ragged edges was found in the antemesenteric border of the ileum, 25 cm. proximal to the ileocecal valve. The entire small bowel was examined from the ligament of Treitz to the ileocecal area and no other perforations or evidence of trauma were found. The perforation was closed and incidental appendectomy was done. After the abdominal cavity was irrigated with copious amounts of saline solution, the abdomen was closed except for Penrose drains that were brought out through a stab wound. The patient was discharged from the hospital nine days after operation and he was well thereafter.

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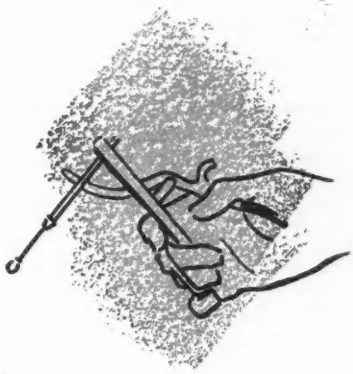

# Intravenous Nutrition

## A Clinical Evaluation of a 50 Per Cent Dextrose in Water Solution, Containing 1 mg. of Hydrocortisone per 100 ml.

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• **A 50 per cent dextrose in water solution, containing 1 mg. of hydrocortisone per 100 ml., was used successfully in 70 patients for intravenous nutritional maintenance and repletion. There were no adverse systemic effects during or following 216 infusions. The only undesirable local reaction was the rare occurrence of pain in the arm when the concentrated solutions were given too rapidly. Glycosuria was minimal if the infusion rate did not exceed 0.85 gm. of glucose per kilogram of body weight per hour, particularly if 50 units of insulin were added to each 550 ml. bottle of 50 per cent dextrose. In patients without significantly elevated serum potassium content, 30 mEq. of potassium chloride, acetate or phosphate was added to each bottle to prevent hypokalemia.**

**Preliminary observations suggest that this new solution may be given safely intravenously, without need for cutdowns or plastic catheters, if the needle is carefully inserted and well immobilized in the arm vein and the duration of the infusion is not too prolonged. Further studies on the effect of such high caloric supplementation plus protein hydrolysates in parenteral nutritional repletion and maintenance are indicated.**

WITH THE PARENTERAL SOLUTIONS and supplements now available, it is possible to treat, prophylactically or actively, most of the common fluid and electrolyte aberrations. However, the severe malnutrition that complicates acute and chronic illness and is often associated with hyponatremia and intracellular potassium depletion, still presents a therapeutic challenge. The major problem is to provide sufficient calories, in addition to protein hydrolysates and electrolytes, to promote repletion of the depleted tissues without administration of excessive fluid to patients who often are already overhydrated, as Moore has emphasized.[12] Unfortunately, concentrated glucose solutions given by intravenous needle, usually produce severe phlebitis and often thrombosis.[16] Therefore, highly concentrated dextrose solutions have not been given intravenously except, as in patients with acute renal failure, via plastic catheters threaded through extremity veins into the vena cava.[18]

Recently, McNair and Dudley[10] emphasized the dangers of thrombophlebitis following this practice, or of septicemia if there is severe infection elsewhere in the body. Moncrief[11] and Hassall and Rountree[7] reported similar clinical experiences. Bogen[2] recently observed that complications developed at the site of the cutdown in 14.5 per cent of 234 cases.

To provide calories without these hazards, fat emulsions for intravenous administration have been developed. Unfortunately, after a few days even the best of these produce febrile or other systemic reactions.[9,13]

Some of the factors contributing to local phlebitis and thrombosis following intravenous infusions, as well as possible prophylactic measures, were investigated by McNair and Dudley[10] who drew the following conclusions:

1. The infusion site plays a role, since significant phlebitis occurred much more frequently in the long saphenous or other leg veins than in arm veins.

2. Venipuncture *per se* is not important because intravenous needles, carefully inserted and well immobilized in forearm veins for as long as 72 hours, produced negligible local trauma.

3. The pH of the solution is not significant, for heat-sterilized dextrose solutions produced no more venous irritation than did filter-sterilized solutions containing less acid.

4. Local clotting may not be of primary importance, since addition of 200 units of heparin per 100 ml. did not reduce the incidence of thrombophlebitis following experimental infusions of more than 24 hours' duration.

5. Local tissue reaction to prolonged infusion was significantly reduced by adding 1 mg. of hydrocortisone per 100 ml. of solution infused.

Polak[15] had previously reported that three patients in hepatic coma showed no clinical evidence of thrombophlebitis during or following infusions of 20 per cent dextrose solution, containing 1 mg. of hydrocortisone per 100 ml., for 32 to 63½ hours. More recently, he and his colleagues[6] reported that

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**TABLE 1.—Sex and Age Range of Patients**

| Sex | Number | Range in Age, Years |
|---|---|---|
| Males | 33 | 23 to 84 |
| Females | 37 | 27 to 89 |

**TABLE 2.—Clinical Data on Patients**

| Clinical Diagnoses | Number of Patients |
|---|---|
| Carcinoma | 18 |
| Cardiovascular disease | 9 |
| Hepatic disease | 9 |
| Blood dyscrasias | 8 |
| Uremia | 6 |
| Peptic ulcer | 6 |
| Ulcerative colitis | 5 |
| Renal shut down | 2 |
| Miscellaneous | 7 |
| Total | 70 |

**TABLE 3.—Frequency of Administration of 50 Per Cent Dextrose and Water Solution with Hydrocortisone**

| Number of Administrations | Number of Patients | Number of Bottles |
|---|---|---|
| 1 | 26 | 26 |
| 2 | 15 | 30 |
| 3 | 9 | 27 |
| 4 | 7 | 28 |
| 5 | 3 | 15 |
| 6 | 4 | 24 |
| 7 | 1 | 7 |
| 8 | 1 | 8 |
| 10 | 1 | 10 |
| 13 | 1 | 13 |
| 14 | 2 | 28 |
| Totals | 70 | 216 |

**TABLE 4.—Mode of Administration of Infusions**

| Mode of Administration | Number of Patients | Number of Infusions |
|---|---|---|
| Intravenous needle | 53 | 150 |
| Intravenous catheter | 7 | 30 |
| Cutdown catheter | 6 | 24 |
| Needle or cutdown | 4 | 12* |
| Total | 70 | 216 |

*20 Intravenous infusions included in I.V. needle column.

in a larger, well controlled series this concentration of hydrocortisone had no effect on the incidence or severity of the local reaction to intravenous infusions administered for more than 24 hours. The latter observation confirmed the reports of Horwitz, Sachar, and Elman[8] and of Carter[3] that the duration of infusion is of prime importance in the pathogenesis of infusion thrombophlebitis.

The present report is concerned with the use of the anti-inflammatory properties of hydrocortisone to reduce the incidence of infusion phlebitis following administration of concentrated dextrose solutions. The data suggest that a solution of 50 per cent dextrose in water, containing 1 mg. of hydrocortisone per 100 ml.,* may be infused slowly with an intravenous needle, without significant local or systemic reaction. As much as 750 gm. of dextrose per day may be given intravenously in only 1,500 ml. of solution without use of a plastic catheter or venous cutdown.

## METHODS AND MATERIALS

Tables 1 and 2 summarize the pertinent clinical and statistical data on 70 subjects. The majority were patients at the Cedars of Lebanon Hospital, but some were seen in consultation at other hospitals in Los Angeles. A total of 216 infusions were given with individual patients receiving from one to fourteen infusions (Table 3). In 53 patients the infusions were administered intravenously with an 18- or 20-gauge needle (Table 4). In seven an in-dwelling polyethylene catheter was threaded through a needle into the vein. In six patients intravenous cutdown was done, generally on a branch of the long saphenous vein. In four patients infusions were given through needles and cutdowns. Thirty-six of the 216 infusions were given through a cutdown or an intravenous catheter.

Although infusions were given more rapidly, at times, to test the patient's subjective and metabolic response to the resulting hyperglycemia, generally the rate was adjusted to provide less than the 0.85 gm. of dextrose per kg. of body weight per hour, which generally may be given to a normal person without producing significant glycosuria.[5] Thus, in four hours a 70 kg. subject would be given 550 ml. of 50 per cent dextrose solution containing 1 mg. of hydrocortisone per 100 ml. Most normal subjects were able to metabolize the 250 gm. of dextrose per bottle with little or no glycosuria. However, to reduce the strain on the beta cells of the islets of Langerhans,[1] 50 units of regular insulin (one unit per 5 gm. of dextrose) usually was added to each 550 ml. bottle. To promote more rapid hepatic deposition of glycogen and potassium in patients with hyperpotassemia, 100 units of regular insulin was added to the first bottle of 50 per cent dextrose solution given each day, and 50 units was added to each subsequent bottle. The total fluid replacement requirements of patients in acute renal failure (estimated from insensible loss plus losses in urine, stool and vomitus) were supplied quantitatively with the concentrated dextrose solution. The 600 to 750 gm. of glucose per day provided sufficient calories to reduce decidedly the breakdown of tissue for energy production.

*Supplied as Molidex by Don Baxter, Inc., Glendale, California.

To each bottle given to patients without hyperkalemia, 30 mEq. of potassium phosphate, chloride or acetate was added, the specific anion being determined by the patient's clinical requirements. In addition, supplements of vitamin B complex, vitamins K and C, magnesium sulfate and calcium gluconate were given, as indicated.

Many of the surgical or malnourished patients were provided with approximately 2,800 calories per day by the following schedules:

1. An initial four-hour infusion of 550 ml. of 50 per cent dextrose with hydrocortisone, containing 50 units of regular insulin, 30 mEq. of potassium phosphate, plus vitamins.

2. A five-hour infusion of 1,050 ml. of a 10 per cent casein hydrolysate (Hyprotigen®) solution containing 7.5 per cent ethyl alcohol, 1 mg. of hydrocortisone per 100 ml., 33 mEq. of potassium, and 84 mEq. of sodium, with 8.2 mEq. of added magnesium sulfate.

3. A final four-hour infusion of 550 ml. of the first solution, containing 8.2 mEq. of magnesium sulfate and 30 mEq. of potassium chloride or potassium acetate, depending on patient's acid-base requirement.

Whenever possible, infusions were given through needles carefully placed into a large vein in the arm. The site of the infusion was inspected frequently to permit correction of faulty placement of the needle or to note any irritation or thrombosis of the vein or infiltration around the vein. After the last infusion was completed, the needle was carefully withdrawn and light pressure exerted at the site of the puncture for several minutes with the patient's arm elevated and the forearm extended. Alternate arms were used on successive days, the available adequate veins being used in rotation. The patients were carefully observed and examined during and 24 hours after each infusion. Any subjective complaints or objective findings were recorded.

Serum electrolytes, blood sugar, and daily urinary dextrose excretions were determined by standard methods on a number of subjects. Pulse, temperature, and often blood pressure were recorded every four hours during and following most infusions.

## RESULTS

A total of 216 infusions were given to the 70 patients (Table 3).

In the 53 patients in whom administration was through a needle, no significant local or systemic reactions were observed. The only complaint recorded (Table 5) was of pain at the site of infusion or along the course of the vein when the infusion rate was too rapid or the vein too small to permit adequate dilution of the decidedly hypertonic solution. In only two instances was the pain severe enough to necessitate discontinuing the infusion. Occasionally, there was some transient soreness at the infusion site. However, in no case did thrombosis or even persistent phlebitis ensue.

**TABLE 5.—*Patient Reactions to Infusions***

| Patient | Subjective Complaint | Subsequent Thrombosis or Phlebitis |
|---|---|---|
| 1. | Local pain during 2 of 3 infusions | None |
| 2. | Pain during infusion | None |
| 3. | Pain during infusion | None |
| 4. | Pain radiating to shoulder | None |
| 5. | Initial pain stopped when infusion was slowed | None |
| 6. | Severe pain requiring cessation of infusion into small vein | None |
| 7. | Pain during 1st hour of 1 of 5 infusions | None |
| 8. | Pain during more rapid infusion | None |
| 9. | Local pain during 1 of 14 infusions | None |
| 10. | Severe local pain requiring cessation of infusion | None |
| 11. | Initial pain stopped when infusion was slowed | None |
| 12. | Initial pain stopped when infusion was slowed | None |
| 13. | Pain in arm after one-liter infusion | None |

In seven agitated or restless patients, the needle slipped out of the vein with resulting subcutaneous infiltration of the 50 per cent dextrose solution with hydrocortisone. However, no inflammation, infection or slough resulted, the only evidence of local reactions being areas of painless ecchymosis.

In contrast, in three of four 550 ml. intravenous infusions of ordinary 50 per cent dextrose in water solution without hydrocortisone there was local pain, thrombosis and phlebitis which subsided uneventfully but obliterated the affected vein.

In seven patients with in-dwelling polyethylene catheters, and in the ten with intravenous cutdown, phlebitis and thrombosis of the veins ultimately developed. Although the house officers felt the vessels tended to remain open longer than ordinarily in patients receiving the 50 per cent dextrose with hydrocortisone solutions, a much larger series will be needed before such a conclusion can be statistically verified.

### Hyperglycemia and Glycosuria

In patients given single slow infusions of 50 per cent dextrose with hydrocortisone solutions without insulin, blood sugar content increased only 70 to 200 mg. per 100 cc. Consequently, only 3 to 16 gm. of dextrose per 24 hours was excreted in the urine. Subsequent infusions produced similar patterns of glycosuria. However, when 50 units of regular insulin was added to each 550 ml. of solution, blood sugar rose less and generally only 2 to 3 gm. of dextrose per day was excreted in the urine even after a number of daily infusions. In patients with either severe hepatic disease or uremia, blood sugar levels tended to be higher, even when insulin was added to the infusion.

TABLE 6.—*Changes in Chemical Contents of Serum and Blood During Treatment of a Uremic Acidotic Hyperkalemic Patient*

| Day | Serum Electrolytes (mEq. per liter) | | | | Blood Urea Nitrogen mg. per 100 cc. | Creatinine mg. per 100 cc. | Hemoglobin gm. per 100 cc. |
|---|---|---|---|---|---|---|---|
| | K | $CO_2$ | Na | Cl | | | |
| 1 (Pre-treatment) | 7.9 | 10.5 | 140 | 117 | 88 | 9.7 | 8.1 |
| 2 | 5.9 | 15.0 | 140 | 112 | 76 | 9.5 | .... |
| 3 | 4.6 | 17.0 | .... | .... | .... | .... | .... |
| 4 | 4.1 | 25 | 140 | 110 | 62 | 9.0 | 9.4 |

**Electrolyte Changes**

Continued infusions without added potassium salts, especially with insulin supplements, were associated with decreases in serum potassium. For example, in one malnourished woman the serum potassium fell to 2.7 mEq. after one infusion and rose slowly when 30 mEq. of potassium phosphate was added to each subsequent bottle. No other significant changes in serum electrolytes were noted, probably because appropriate sodium, calcium and magnesium salts were added, as indicated above, for patients who were receiving all their nutrition parenterally.

**Uremia with Acidosis and Hyperkalemia**

In uremic and other hyperkalemic patients, serum potassium levels fell promptly and progressively following infusions of 50 per cent dextrose with hydrocortisone. Table 4 presents data on a typical patient, a 32-year-old man with uremia resulting from a congenitally fused kidney and chronic pyelonephritis. On admission, he appeared moribund, having severe oliguria, respirations of 65 per minute due to pronounced acidosis, and the blood contents typical of severe uremia, acidosis and hyperkalemia (Table 6). He was given a four-hour infusion of 550 ml. of 50 per cent dextrose in water with hydrocortisone, supplemented with 100 units of regular insulin and 90 mEq. of sodium lactate, followed by an infusion of 1,000 ml. of 10 per cent dextrose in water, supplemented with vitamins B and C, 40 units of regular insulin and 90 mEq. of sodium lactate. By the next morning, serum potassium had fallen from 7.2 to 5.9 mEq. per liter and the carbon dioxide content rose from 10.5 to 15.0 mEq. per liter. That day another liter of the augmented 10 per cent dextrose in water was given, preceded and followed by 550 ml. infusions of the 50 per cent dextrose with hydrocortisone plus insulin and sodium lactate, and the serum electrolyte situation improved further. The next day only one 550 ml. bottle of the concentrated dextrose with hydrocortisone was given, followed by two units of blood and a liter of the augmented 10 per cent dextrose in water solution. By the following day, when the patient was well enough to be discharged home, the electrolyte contents were essentially normal.

**Effects of Hydrocortisone**

No evidence of any adverse effects of small amounts of hydrocortisone administered were observed in any patients. The largest daily dose was only 21.5 mg. when two bottles of the concentrated dextrose solution and one liter of the supplemented protein hydrolysate solution were given.

## CLINICAL RESPONSE

In patients with chronic uremia or acute renal failure, the clinical response was excellent with rapid correction of hyperkalemia and acidosis and lowering of the blood urea nitrogen content. In malnourished patients, even those with metastatic malignant disease, improvement was noted following the increased caloric and nitrogen intake. After five or six days of vigorous parenteral nutrition, many patients regained appetite, began to eat and showed pronounced increase in vigor. No adverse clinical responses were observed.

It should be noted that, although this report deals only with 216 infusions, almost 500 bottles of 50 per cent dextrose with hydrocortisone have been administered at this hospital. The present series includes only those cases in which adequate observations were made. However, no significant reactions other than occasional transitory local soreness have been noted in the larger series. Moreover, the use of the new solution on all the clinical services has been steadily and spontaneously increasing, whenever adequate parenteral nutrition is needed.

## DISCUSSION AND CONCLUSIONS

The present data demonstrate that properly prepared 50 per cent dextrose in water solutions, containing 1 mg. of hydrocortisone per 100 ml., may be given intravenously through an ordinary needle at a rate providing 0.85 gm. of dextrose per kg. of body weight per hour, without significant local or systemic reaction. Although limited control studies tend to confirm the widespread clinical impression that similar administration of 50 per cent dextrose solutions without hydrocortisone generally produces decided venous irritation, and often phlebitis or thrombosis or both, a larger control series is neces-

sary to provide statistical support for this conclusion. However, the resulting discomfort and danger to the patients made it difficult to justify the continued use of the control solutions in this study, particularly in view of the therapeutic success being achieved with the solutions containing 0.001 per cent hydrocortisone.

Fortunately, the maximal rate of infusion associated with minimal glycosuria is well tolerated by most patients. However, at times an even slower rate may be required to avert local pain due to the hypertonicity of the solution. To reduce venous irritation, the 20 or 22-gauge needle should be carefully inserted in a large vein in the arm, firmly immobilized, and removed in less than 15 hours. The duration of infusion, particularly, must be carefully controlled, as has been emphasized by many investigators.[3,6,8,10,16]

Despite these limitations of rate and duration of administration, it has been possible, in 13 or 14 hours, to give infusions providing 550 gm. of dextrose, 100 gm. of casein hydrolysate, 75 ml. of ethyl alcohol, 93 mEq. of potassium, 84 mEq. of sodium, and 16.4 mEq. of magnesium in a total volume of 2,150 ml. containing only 21.5 mg. of hydrocortisone. Such parenteral therapy, supplying protein hydrolysate and electrolytes plus 2,800 calories in only 2,150 ml. of solution, will not only adequately maintain well-nourished medical and surgical patients but may also permit repletion of severely malnourished subjects by promoting utilization of the nitrogen administered.

As emphasized by Pareira and Somogyi[14] and Spencer and Beal,[19] improved appetite often follows intravenous administration of sufficient dextrose to correct the ketosis of starvation. This factor plus the increased vigor following partial nutritional repletion may explain the decided improvement in oral intake observed in many patients in the present series after five or six days of adequate parenteral therapy. Wider application of these solutions is required to determine if the anticipated salubrious effect on the morbidity following extensive gastrointestinal operations, trauma, burns, comatose states and other debilitating clinical conditions will be achieved.[12,16,19]

In patients with acute renal failure, infusion of 1,000 to 1,500 ml. of concentrated dextrose with 0.001 per cent hydrocortisone and the proper amount of insulin not only provides sufficient calories for metabolic maintenance, thereby reducing tissue breakdown, but also promotes deposition of potassium with glycogen, thus decreasing hyperkalemia. Preferably, the infusion should be given intermittently, alternating veins from arm to arm. If continuous infusion is needed, a different vein should be used every 12 to 14 hours. As Salisbury[17] recently noted, metabolism in profoundly uremic patients may be so distorted, at times, as to impair utilization of carbohydrate. In these circumstances concentrated dextrose infusions may produce increasing hyperglycemia which is unaffected by concurrent administration of large doses of insulin.

As indicated above, a larger control series will be required to determine whether properly prepared 50 per cent dextrose solutions without hydrocortisone may be given without venous trauma if the precautions described in this study are taken. Spencer and Beal[19] reported that phlebitis was uncommon following infusion of 25 per cent dextrose plus 5 per cent alcohol in water solutions through a 22-gauge needle carefully placed in a vein. They suggested that perhaps the hazard of phlebitis due to infusions of hypertonic dextrose solutions has been overemphasized in the past. However, the absence of significant local reaction to accidental subcutaneous infiltration of the 50 per cent dextrose solutions with hydrocortisone is consistent with the anticipated anti-inflammatory properties of the steroid.[15] It is in striking contrast to the local redness and soreness following subcutaneous infiltration of other solutions without hydrocortisone.[16] The failure of the steroid to reduce the incidence of phlebitis and thrombosis following infusions of more than 24 and 48 hours' duration[6] is not surprising. Under these conditions, the time-intensity factor probably far exceeds the physiological-pharmacological potential of the hydrocortisone.

In conclusion it should be reemphasized that these observations are preliminary. However, the results observed in this and in other[4] clinics suggest that 50 per cent dextrose in water solution, containing 1 mg. of hydrocortisone per 100 ml., may be given intravenously to provide calories in a limited fluid volume. Further studies on the clinical and metabolic effects of such treatment in the management of patients with acute and chronic renal failure, and for nutritional maintenance or repletion of medical patients, as well as surgical patients, preoperatively and postoperatively, are in progress.

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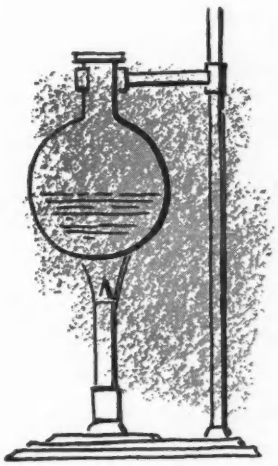

# Wanted: A Biochemical Test for Schizophrenia

RONALD R. KOEGLER, *M.D.*, EDWARD G. COLBERT, *M.D.*, and SAMUEL EIDUSON, *Ph.D.*, Los Angeles

• **Recent discoveries and refinements in technique in the field of biochemistry have led to renewed interest in the idea that a test can be developed for the diagnosis of schizophrenia. Studies directed toward that goal have included investigations of biological amines, carbohydrate metabolism, epinephrine metabolism, serotonin, taraxein and ceruloplasmin. No conclusive evidence of any biochemical abnormality in schizophrenia has been found. Although careful studies in adults have failed to confirm a theory that ceruloplasmin levels are abnormally high in schizophrenia, the surmise that it might be true in schizophrenic children was investigated, since constitutional factors seem to be very important in this condition. Thirty-four schizophrenic children and a control group of 13 "behavior problem" children were investigated. No difference was found between the two groups in serum content of copper, ceruloplasmin or ascorbic acid.**

THE IDEA of a single laboratory test that would accurately determine or even strongly suggest a diagnosis of schizophrenia is an attractive one. The quest for such a test has stimulated research in this field for many years, and much worthwhile information about mental illness has been obtained.

The concept of biochemical changes as a cause for schizophrenia has been given a good deal of attention since the early years of the Twentieth Century. In a less sophisticated form it has appeared in many earlier civilizations. The "humors" of the ancient Greeks are prototypes of modern concepts of biochemical forces.

> "As long as the brain is at rest, the man enjoys his reason, but the depravement of the brain arises from phlegm and bile, either of which you may recognize in this manner: Those who are mad from phlegm are quiet, and do not cry out or make a noise; but those from bile are vociferous, malignant, and will not be quiet, but are always doing something improper."
>
> —Hippocrates, *On the Sacred Disease*[13]

In the early 1900's a number of investigators announced the discovery of biochemical abnormalities in schizophrenia, based on their studies of metabolites in the blood and urine. Similar studies were published with varying degrees of enthusiasm in the 20's and 30's. This work was reviewed in some detail by McFarland and Goldstein,[25] and more recently by Kety,[18] all of them concluding that the reported observations were inconsistent and inconclusive. Recent improvements in laboratory techniques have led to renewed interest in schizophrenia as suitable for biochemical research. Present-day biochemical research tends to follow many diverse paths, depending on the hypothesis of the investigator, but several concepts have come to have more than passing attention.

### Biological amines

Recently there has been increased interest in hallucinogens that are related to biological amines, and this has turned attention anew to amine metabolism in schizophrenia. New research has been made feasible by the development of paper chromatographic techniques, and a great number of studies of the urine of schizophrenic patients have been reported. Among the reported observations are differences in excretion of aromatic compounds,[26] higher concentrations,[26] presence of abnormal amines[11] and the absence of a normally occurring substance.[9] These conflicting reports have been criticized on the grounds that dietary factors were not controlled and statistical analysis was unsound.[27]

### Carbohydrate metabolism

Investigators have reported such varied observations as decreased cerebral blood flow and decreased cerebral glucose consumption,[7] abnormal glucose tolerance[22] and reduction in the blood gluathione index.[23] Other studies have cast doubt on these hypotheses,[5,36] and the concept of such generalized defects in schizophrenia does not seem too attractive to most investigators.

### Epinephrine hypothesis

Several studies have purported to show that in schizophrenia there is an abnormal metabolism of epinephrine and that the hallucinogenic properties of such epinephrine derivatives are related to many of the symptoms in schizophrenia.[14,29] Hoffer[14] suggested that adrenochrome or adrenolutin might be the guilty substance. Attempts to confirm these re-

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ports in carefully controlled studies have failed.[15,35] The epinephrine hypothesis is an attractive one, but the evidence for it is scant.

### Serotonin

The finding of serotonin in the brain and the discovery that lysergic acid diethylamide (LSD) could block the effects of serotonin in smooth muscle[37] led to interest in the possible relationship of serotonin to schizophrenia. Interest was further stimulated by the finding that reserpine causes a decrease in the content of serotonin in the brain.[34] There has been no satisfactory theory that can explain the apparently mutually exclusive findings, let alone relate them to schizophrenia. This is one of the areas in which much current work is being done, and a great deal is being learned about physiologic, pharmacologic and biochemical features of the brain, even if no test for schizophrenia results.

### Taraxein

Heath and coworkers at Tulane[20] reported the finding of a substance in the serum of schizophrenic patients, perhaps a form of ceruloplasmin, which they called "taraxein." It was supposedly capable of inducing a schizophrenic-like state when injected into normal volunteers and of causing various changes in laboratory animals. Other investigators have been unable to isolate this mysterious substance.[30]

### Ceruloplasmin

Ceruloplasmin is an extremely interesting protein substance. Isolated by Holmberg and Laurell in 1947,[16] and named "ceruloplasmin" because of its deep blue color, it tightly binds almost all the copper in the body. A relative deficiency of ceruloplasmin plays an important role in the etiology of Wilson's disease.[32]

It has been known since 1951 that ceruloplasmin could oxidize p-phenylenediamine as well as other amines.[21] There had been a few reports stating that there was increased ceruloplasmin and copper in the blood of schizophrenic patients[19,28]; however, little interest was shown until a 1957 report by Akerfeldt[3] of an increased oxidation of n.n-dimethyl-p-phenylenediamine by the blood of schizophrenic patients. That report led to a surge of interest in this new "test" for schizophrenia. It was noticed that ascorbic acid levels could affect the results; however, Abood[2] developed a modification which was little affected by ascorbic acid. The first reports tended to confirm the differences in schizophrenia, but recent investigations have failed to show this phenomenon.[4,31]

## PRESENT STUDY

We were interested in the relationship of ceruloplasmin levels to schizophrenia. However, one of the problems in doing research in schizophrenia is that there is probably a diversity of etiologic factors. When Bleuler described the "group of schizophrenias," he was referring to just this. Therefore, it is easily possible to overlook abnormalities when the group of schizophrenic persons studied is not sufficiently homogeneous. It is difficult, in our present state of etiologic knowledge of schizophrenia, to determine when we are working with a homogeneous group.

It was our thought that if we took a group of seriously ill patients in which the onset of the disease was at approximately the same age we might have at least a degree of similarity. For this reason we chose to study a group of schizophrenic children in whom definite signs of the disease had developed in the first three years of life. It has been shown that many of these children grow up to be adult schizophrenics of the most regressed type.[6]

Our study group consisted of 34 schizophrenic children and our control group was 13 children with behavior problems. All were in the children's unit of the same state hospital. Blood was drawn from each child and serum copper, ceruloplasmin and ascorbic acid levels determined. The methods of diagnosis are described in detail in a previous publication.[10]

The Abood modification of the Akerfeldt test was used to determine ceruloplasmin levels.[1] Copper in the serum was measured by the Gubler method.[12] The Schaffert and Kingsley technique[33] was used to evaluate ascorbic acid in the serum.

We found no significant difference between the two groups in serum copper, ceruloplasmin or ascorbic acid levels. This is in keeping with McDonald's[24] observation with regard to adult schizophrenics; a high ceruloplasmin level is not characteristic of schizophrenia, and does not occur more frequently in persons with this disease than in normal persons, given an adequate diet and normal ascorbic acid levels. Brown[8] recently noted high ceruloplasmin content in 30 per cent of non-schizophrenic children ill with other diseases.

## DISCUSSION

To the extent that the patients studied represented a homogeneous group, the fact that no abnormality of ceruloplasmin levels was observed has some significance. We attempted to divide the group into autistic and non-autistic schizophrenics, according to Kanner's criteria,[17] but this had to be abandoned because the children in the study tended to have some characteristics of both conditions.

However, it is very likely that in some of these very similar children the etiologic factors in the disease were quite varied. In one child genetic factors might be quite important, while in another intense parental rejection and neglect might be paramount. Even though in clinical state the patients were similar to each other at the time of hospital admission, it is plausible that the biochemical patterns differ both quantitatively and qualitatively.

It would seem, then, that we are limited in our search for a biochemical test for schizophrenia by lack of knowledge concerning both the causes and the categories of the condition. Of course there is still hope that by more subtle biochemical techniques or new approaches we may be able to devise a test that will detect chemical changes occurring in all forms of schizophrenia, regardless of the etiologic differences, and not occurring in other diseases. All that we can say at this time is that the determination of serum ceruloplasmin is not such a test.

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#### DISCUSSION by ALLEN J. ENELOW, M.D., Los Angeles

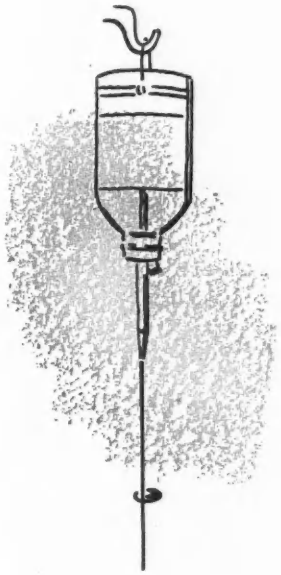
The authors have made an excellent and comprehensive review of the present status of biochemical research in the heterogeneous group of patients called schizophrenics. However, the title of their paper implies a specific disease—even though the authors express their doubt about there being a specific disease, schizophrenia, in their paper. When I think of the term *schizophrenia*, I get a certain picture in my mind. I really doubt that this picture is exactly the same as the one any other psychiatrist gets when he thinks of schizophrenia. If we were to attempt to settle these differences by coming to some agreement on what he and I consider to be crucial to the diagnosis of schizophrenia, we might or might not be able to do so. But even if we could do so, there would still be a sizable group of patients which one of us and not the other would want to call schizophrenic.

This statement seems to me to describe the dilemma of the investigator who wants to study schizophrenia. The authors have gotten around this to some extent by limiting their study to a group of children who became seriously psychotically ill at an early age. As it happens, they got negative results. But, even if they had gotten positive results, nothing would have been done to resolve the dilemma of psychiatry today about the serious psychotic illnesses, whatever we call them.

I propose that the term schizophrenia is a disadvantage to the investigator. It might be more fruitful to stop looking for similarities in this large and ill-defined group of patients and begin to look for differences. This possibility did not

escape the authors who in fact state that even though the clinical pictures of their subjects were similar, it is plausible that there might be considerable differences in the biochemical picture. I believe it unlikely that chemical changes or any other kinds of changes will be turned up which occur in all patients currently labeled schizophrenic. I feel, rather, that the most fruitful approach to this problem would be to study, from as many different avenues of approach as possible, the differences between all of these patients who are now called schizophrenics. Such studies should be biochemical, behavioral, and sociological. Many such studies are going on, of course, as the authors mention. But there is a tendency to pay lip-service to the ill-defined nature of this group of patients and then act as though there were, in fact, such a disease.

My own particular notion about so-called schizophrenic symptoms is that they are a non-specific group of symptoms—a sort of final common pathway, as it were, for a variety of disturbances. The underlying disorder may have a fairly good prognosis in some instances, and a grim one in other instances. Some of these disorders may turn out to be curable, others not. Some may even be preventable, when we know more about them. But the group of symptoms which currently lead to the diagnosis (often disputed) of schizophrenia may well be as non-specific as fever, flushing, chills and headache.



# Casts of the Vagina as a Means of Evaluating Structural Changes and Treatment

KENNETH F. MORGAN, JR., M.D., Los Angeles

• A method of making three-dimensional molds of the vagina with the materials used by dentists for oral impressions gives considerably more information about normal and abnormal anatomic features than other methods of study, including direct viewing, palpation and contrast x-ray studies. The molds are of value in the demonstration of anatomic patterns in relation to normal and abnormal functions, in evaluation of surgical and other therapeutic techniques, in teaching, and in explaining the abnormalities and the aims of treatment to patients.

The use of such molds is being applied to the study of pelvic prolapse, urinary stress incontinence, sexual function of the vagina and birth injuries.

ACCURATE TREATMENT of pathologic structural changes of the vagina and its pelvic environs and supports requires a determination of physiologic and anatomic conditions in individual patients for purposes of comparison with what is known of physiologic norms. The present report is of a method of making casts of the shape of the vaginal vault to determine general norms and the departures from normal in disease states. Made serially over a period of treatment, the casts are a useful record of progressive changes by which the effectiveness of therapy can be evaluated.

Current concepts of vaginal anatomy have been arrived at by the classical methods of the anatomist —examination of living subjects, dissection of cadavers, x-ray visualization and histological techniques. Vaginal surgical procedures have been based on observation of anatomic structure with the patient in the lithotomy or supine position and on trial and error, with emphasis on fasciae and ligaments. Sexual physiology of the vagina and its relationship to fertility, to general pelvic function and to female psychology is clouded. The exact mechanisms of urinary control remain illusive and subject to considerable controversy.

Studies by several investigators who gave primary consideration to function have somewhat modified anatomic concepts. Dickinson made careful measurements and drawings from a large number of patients, correlated them with histories and reported them in his atlas published in 1933.[1] He attempted the taking of vaginal impressions on wax cylinders in order to demonstrate muscular patterns. Van de Velde,[10] through careful examination and history-taking, made contributions toward true understanding of sexual function in women. Kegel,[2-9] in his 30-year study of the pubococcygeus muscle, further advanced the knowledge of vaginal and pelvic anatomy in relation to function.

The research here reported was done with Dr. Arnold Kegel, emeritus professor of gynecology at the University of Southern California. It was carried out in the pelvic relaxation clinic at the Los Angeles County General Hospital. Our interest in the physiology of the pelvis as a neuromuscular unit, and in particular the pubococcygeus muscle system and vagina, led to a search for a better method of studying the anatomic features and pathologic variations of the vagina in living subjects. We needed a way to show the actual effect of pubococcygeus muscle exercise in simple pelvic relaxations and urinary stress incontinence, to clearly demonstrate the kinds of injury to the vagina occurring at the time of delivery in order to correlate these injuries with their far-reaching consequences, and to evaluate plastic surgical procedures in the vagina.

Heretofore, findings have been reported in terms of clinical impressions, the patient's reports and perineometric readings.

Our first attempts at making molds of the vagina fortunately coincided with the development of a new material for taking impressions in the oral cavity. It is a powder which forms a rather thick paste when mixed with water. The paste solidifies in about two minutes, forming a resilient, rubbery mold which records detail with extreme accuracy. The material is chemically inert in the vagina and soft enough to permit removal without injury to the patient. The cast obtained is an accurate three-dimensional negative.

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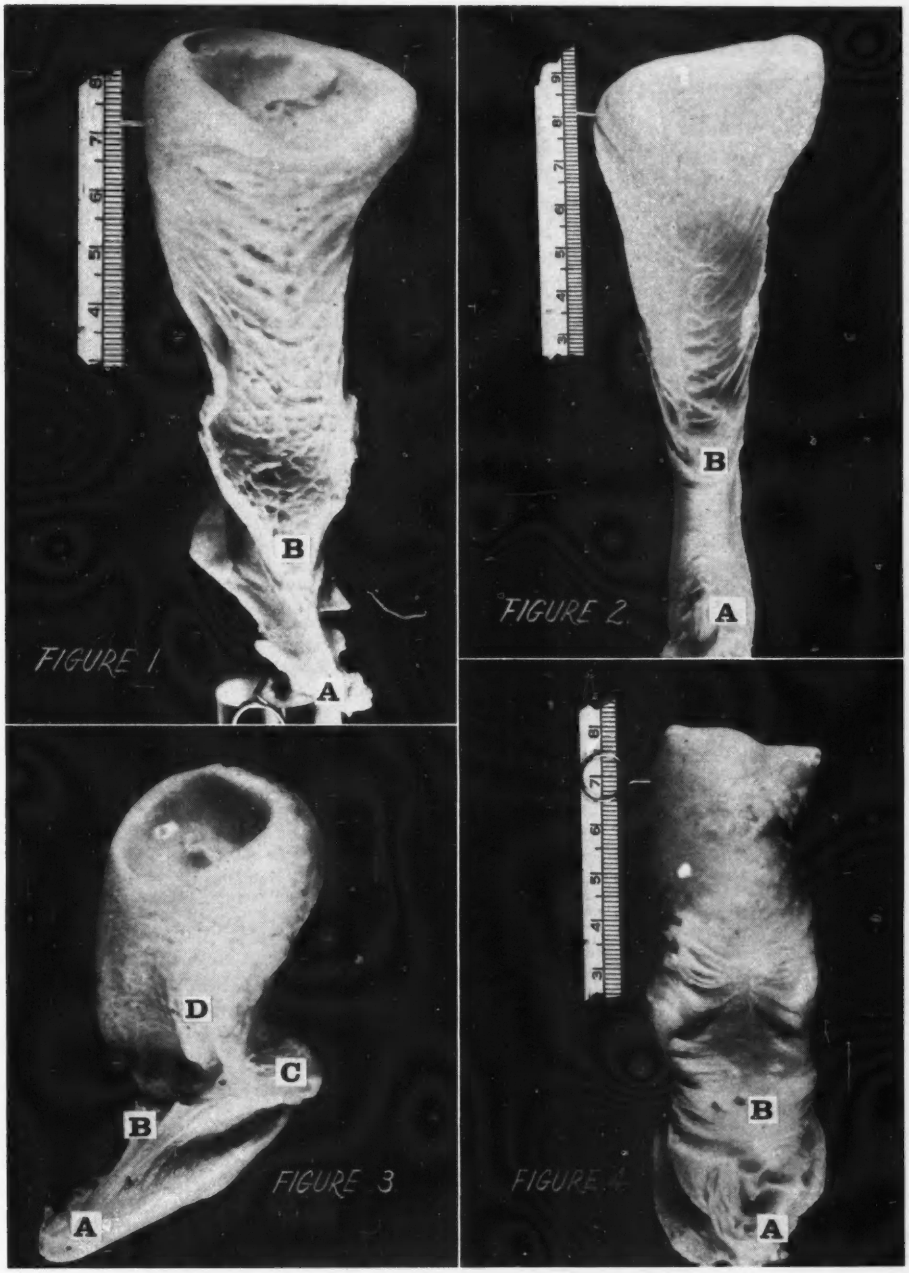


Plate I.—A, Clitoris; B, urethral meatus; C, indication of defect in pubococcygeus muscle; D, indication of scars running to the bladder base. The scales shown are in centimeters. *Figure 1:* Mold of normal vagina, showing symmetrical rugal pattern, muscular constriction of long middle segment and the cervical impression in the wide upper third of the vagina where muscular support is absent. *Figure 2:* Three months after vaginal hysterectomy and anterior and posterior repair. All pelvic functions in the patient were normal. *Figure 3:* Mold of a patient with left mediolateral episiotomy injury, showing indication of a defect in the pubococcygeus muscle and scar tissue running to the region of the bladder base (which caused urinary stress incontinence). The muscular segment is shorter than in a normal vagina. *Figure 4:* The mold shows scarring, distortion and complete loss of rugal pattern (following vaginal hysterectomy and anterior and posterior repair). The patient had urinary incontinence. The muscular segment is replaced by rigid scar tissue and the vaginal vault narrowed.

## TECHNIQUE

The procedure is carried out as follows: After the vagina has been lubricated, the casting material is mixed according to directions and is then drawn into an injector made of a 7/8-inch polyethylene tube with lucite plunger and a rubber washer. The mixture is deposited in the vagina by inserting the injector to the vaginal vault and withdrawing the tube while holding the plunger stationary, care being taken not to force the paste lest pressure change the shape of the organ. While a pad is held against the vaginal orifice just firmly enough to prevent escape of the paste, the patient is put into whatever position is suitable for purposes of the examination—lithotomy, standing, sitting or prone—and is kept in that position for the two minutes necessary for the paste to solidify. Usually the cast is easily removed by pulling gently on it as the patient bears down. But if it is not readily delivered, perhaps owing to a large vault and narrow introitus, the cast can be broken up or can be pushed out by pressure applied rectally.

After inspection photographs are made for permanent records to attach to the patient's chart. Since the casting material shrinks as it dries, the casts themselves are useless as permanent records and should be discarded.

An unexpected advantage that became apparent in the course of our experiments is that it is very simple to make molds with the patient standing. Since this is the position of maximum stress, these molds were more informative than those that were made with the patient in the usual lithotomy position. In normal patients we found no difference between molds made in the lithotomy position and those with the patient standing, but in abnormal situations all degrees of sagging and distortion were shown in casts made with the patient standing.

The molds are a valuable aid in teaching detailed examination of the vagina. After a student examines a patient, a mold is made for him to study, and he then reexamines the patient with the advantage of having seen what he is expected to palpate.

Giving instructions and explanations to a patient is made much easier if she can be shown the differences between a mold of her vagina and one of a normal vagina. The reasons for contemplated surgical procedures can be demonstrated clearly. Letting a patient know that molds will be made from time to time to show the progress of therapy can be a valuable inducement to full cooperation.

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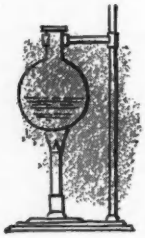

# Industrial Visual Screening

## Advantages and Disadvantages of Various Instruments

BYRON H. DEMOREST, *M.D.*, and JOHN A. BERG, *M.D.*, Sacramento

• **The ideal test for visual screening is one which is easily performed by a technician with limited training, inexpensive and not time-consuming, easily understandable by all applicants, and one which will correspond generally with a more thorough examination by an ophthalmologist. The ideal screening technique should test accurately those functions needed for any particular occupation. The visual screeners now in great preponderance have certain advantages for ease and are generally acceptable for approximating the visual acuity. Visual screeners do not accurately test the astigmatic applicant, and they have not proven their value in testing depth perception and color vision. The use of the Harrington Flocks Screener is recommended for testing the visual field. The use of the Verhoeff Steropter for depth perception and the American Optical pseudoisochromatic plates for color testing is recommended when these tests are needed.**

**The old Snellen test cards, or the projector chart for measuring distance vision, and the test cards for measuring near vision are often much more reliable than are the visual screeners.**

THE DETERMINATION of the visual status of job applicants is now looked upon as one of the most important items in the pre-employment physical examination. Many investigators[2,4,12,13,14] have stressed the merits of examining the eyes of prospective employees and have suggested that industrial workers should have visual screening at regular intervals throughout their working years. For many jobs, sharp eyesight is a prerequisite. In others, if there is an industrial accident or question of loss of vision from any industrial situation, the value of having a visual base line is great. Follow-up examinations during the span of employment are important to both the employee and the employer. These examinations serve to protect the employee from accidents due to visual impairment, and their value in preventative medicine should not be underestimated.

Eye problems of industry are so prevalent that there are now a number of ophthalmologists who devote the greater part of their practice time to industry. Kuhn[10] has written an excellent book on the subject—*Eyes and Industry*, which should be consulted by anyone planning to establish an industrial visual program.

Not industrial ophthalmologists primarily, the authors of this communication had their interest in the problems of visual screening brought to a focus about two years ago when Dr. Christopher Leggo, Medical Director for the State of California Personnel Board, became involved in establishing visual standards for state employees. His problems were identical to those of any large industrial situation where a great diversity of job classifications exist. Basically the main problems were: Establishing visual standards for the various classes of employees, selecting the tests for measuring visual functions and selection and training of personnel to apply the tests.

This paper will be predominantly concerned with the selection of tests for visual function and the limitations of some of the testing equipment.

When selecting equipment for measuring vision, the examiner should first carefully consider the classes of job applicants and the various visual categories into which they fall. How thorough an eye examination must be certainly depends upon the needs of each position. Cooper[4] wrote an excellent review of rationales for the classification of subjects visually, and suggested means of categorizing employees. Generally these categories are three:

1. General classes of employees. These include most office workers, etc.

2. Special classes in which a high visual standard is required. These include security positions or those in which particularly fine visual skills are needed to operate special equipment.

3. Special classes in which very poor vision is tolerable.

The visual functions which might need testing are the following: Central visual acuity at distance; central visual acuity at near; the visual field; color discrimination (not needed for most); depth perception (need limited to a very few select applicants); refraction, tonometry, and check for progressive ocular disease.

A trained technician is easily able to record results of testing for the first five requirements. However, the industrial ophthalmologist is needed for

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more complete examinations and he should be available, when necessary, to interpret the tests done by technicians. One ever-present problem in vision screening is that applicants are all too frequently given false impressions by a technician who overzealously wishes to impress with scientific knowledge. A visual screening program should never be acknowledged as an eye examination, and in fact effort should be made to keep the applicant from getting such an impression. If the technician is properly instructed to record but not to interpret visual function, such problems do not usually arise.

Various instruments are used in the measurement of certain visual skills:

**Distant Vision**

In general three methods are available for the evaluation of distant vision. These are the wall chart, using Snellen figures, the projecto-chart, using Snellen figures, and the various screening tests that employ test patterns and letters.

*Snellen Letter Wall Chart:* This chart is made up of rows of letters of different size, marked with the number of feet at which distance each line should normally be read. In spite of certain weaknesses, this method has survived for almost 80 years, and all other means for the evaluation of distant acuity use the Snellen letter as a standard.

The advantages of this technique are that it is inexpensive and readily available. It is usually accurate enough to meet the needs for the casual examination of most job applicants. To date there is no readily available device that might replace its general use.

The disadvantages of the wall chart are: The letters may be easily memorized, and there are schools in this country where such charts are committed to memory. In practice the examiner must allow a 20-foot space for proper use, the lighting should be standard, and the extraneous interferences should be limited. The chart needs to be kept clean, and occasionally replaced. The technician must be instructed to prevent cheating by the applicant. Testing with this chart may be augmented by the use of the Lebensohn Unmemorizable Chart.

*The Projector Chart:* For charts projected onto a screen for the subject to read, the time-honored Snellen system is used, but the letters presented may be altered, thus preventing memorization. The disadvantages are the need for the proper set-up for projection and the careful training of an operator to use the apparatus correctly. This device is used by most ophthalmologists.

*The Visual Screeners:* Several large and impressive pieces of equipment are available for screening the vision of job applicants. The more commonly recognized screeners are the sight screener, manufactured by the American Optical Company, the Orthorater, by the Bausch & Lomb Optical Company, the Telebinocular by the Keystone View Company, and the Vision Tester by the Titmus Optical Company. Each of them presents a very impressive battery of tests, and it is easy to gain the impression that any of the procedures is a complete analysis of visual function. This is far from the case. It should be stated, however, that research work in the mechanics of vision testing is continuing, and we can expect modifications or alterations in some, if not all, of the instruments now available.

These visual screeners may be recommended with certain reservations. The advantages are that distant vision may be fairly accurately tested in a limited space, the test is easily done by a technician, and it is made under standard conditions. In addition the test is so arranged that the applicant cannot cheat, and the results can be determined quickly.

One disadvantage is that persons with very poor vision due to astigmatism may be able to do well on a screener; and recently valid cases have been reported in which subjects with myopia have done better on the visual screener test than they do with the standard wall tests for distance. In addition the screeners cost much more than wall charts. If the screener is used with these reservations, and if the results do not need to be absolutely accurate, it is acceptable as a distance vision test for industry.

**Near Vision**

The test for near central acuity may be done in two ways, either with the various near testing cards and charts or with a visual screener. The use of a near test card is excellent if the test is done correctly, which is not difficult even for a person with limited experience. It must be done with a clean card and the card should be unmemorizable.

Visual screeners, when used for testing near vision, have many of the advantages and disadvantages that they have in tests for distant vision.

**The Visual Field**

The third most important aspect of the visual status is the visual field. This particular function is not tested by means of standard equipment on any of the industrial visual screeners now in existence, and hence the value of this function has been all too often overlooked. The peripheral vision is essential for the proper driving of a motor vehicle, a fact that has received attention from the Motor Vehicle Department in California[1] and probably will receive more. If a person's field of vision is narrowed to 40 degrees rather than the normal binocular 180 degrees, driving might be extremely hazardous. The most accurate determination of the visual field is through use of the perimeter and the central tan-

gent screen. In recent years, however, Harrington and Flocks[6] have presented an entirely new instrument which has obvious advantages in industry, for the detection of gross visual field defects. New models are now being perfected[5] which will make the screener less liable to "false positives" results and also make the use of the screener more simple in operation.

**Depth Perception**

The old Howard Dohlman test that was widely used in the armed forces during World War II has been largely abandoned because of its inaccuracy. The more recent compact Verhoeff Steropter provides a simple test which is preferred by the armed forces and ophthalmologists alike. This test is inexpensive, simply done and foolproof; and in the *few* cases in which stereopsis is vital to the performance of any given occupation, this test is highly recommended.

It must be emphasized that depth perception, or stereopsis, is usually not at all important in the majority of job situations. Kirshberg[9] found no relationship between depth perception and flying ability, as shown by an analysis of data gathered on graduates from the pilot training program of the British Commonwealth Air Training Plan during the last war. Cibis[3] said that linear perspective is much more reliable than depth perception in flying and landing airplanes. A similar situation exists in operating automobiles and other moving machinery.

The loss of depth perception due to amblyopia is therefore all too often over-emphasized, for most persons compensate for this loss. Leggo,[11] in emphasizing the need for employment of one-eyed patients, stresses the unimportance of binocular depth perception.

The use of the various vision screening devices to test depth perception has not been successful, and their use to check this particular function is contraindicated. This has been discussed by Hogan.[7]

**Color Discrimination**

When indicated, color discrimination is best tested by the Ishahara test, or, as it is more commonly called in this country, the A-O pseudo-isochromatic color test. Uniform lighting conditions and an understanding of the test by the applicant are important. The visual screeners have not been useful in evaluating this facility. Again, color discrimination is not always of vital importance.

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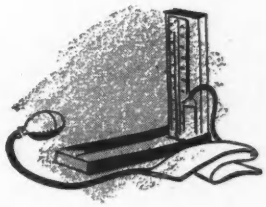

# CASE REPORTS

## Volvulus and Gangrene of the Cecum in a Patient with Ulcerative Colitis

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THE SURGICAL COMPLICATIONS of ulcerative colitis are numerous and varied in nature. Instances have been reported of abscess,[1] peritonitis,[1] fistulae,[2] perforation,[3] hemorrhage[4] and stenosis.[5] A report of a case in which gangrene of the cecum also developed is presented here both because of its rarity and because it points up the necessity of constant observation in cases of ulcerative colitis.

### REPORT OF A CASE

The patient, a 66-year-old white woman who had been receiving therapy for ulcerative colitis for some seven years was examined because of sudden onset of severe cramping pain in the lower abdomen about two hours after she had eaten lunch. She felt nauseated but had not vomited.

The patient appeared to be acutely ill and dehydrated. The pulse rate was 100, and the blood pressure was 120/60 mm. of mercury. The rectal temperature was 101° F.

The abdomen was decidedly distended and exquisite tenderness to palpation was noted throughout the entire abdominal wall. No peristalsis could be heard.

Leukocytes numbered 50,600 per cubic millimeter —with neutrophils 94 per cent (10 per cent stab forms) and lymphocytes 4 per cent. The hemoglobin content was 14 grams per 100 cc. and erythrocytes numbered five million per cubic millimeter. The results of urinalysis were within normal limits.

X-ray films of the abdomen showed a pronounced distention of intestinal loops. The impression was that the involved dilated loops were those of the terminal small intestine and ascending colon. No gas pattern could be made out in the areas of the descending colon or rectum. The roentgenographic diagnosis was intestinal obstruction involving the terminal small bowel and ascending colon. The possibility of a volvulus was considered.

During the seven years the patient had been under treatment for ulcerative colitis (therapy consisting of special diets and supportive care) she occasionally had had episodes of bloody diarrhea which abated rapidly. At no time had the possibility of surgical intervention been considered, the general health of the patient having continued to be good.

Laparotomy was performed approximately two hours after the onset of symptoms. A right pararectus incision was made and a tremendously dilated cecum came immediately into view. There were extensive gangrenous changes throughout the entire cecal wall. The inferior pole of the cecum was adherent to the pelvic floor in the hollow of the sacrum. Complete volvulus of the cecum and terminal 12 inches of ileum had occurred, with the fixed adherent cecal wall acting as a point of rotation for the volvulus. The small bowel proximal to the volvulus was only moderately distended. The ascending colon above the volvulus was flat and completely collapsed. Apparently, small bowel content was entering into the cecum and was prevented from flowing into the ascending colon by the volvulus. Furthermore, the ileocecal valve was obviously competent and the intestinal content could not escape backward into the terminal ileum. As a result, the cecum had become progressively more distended, with resultant vascular changes in the cecal wall. The cecum was easily separated by finger dissection from its point of adherence on the pelvic floor. When the cecum was freed, there was an immediate resolution of the volvulus, and the ascending colon filled with gas and intestinal content. The terminal ileum was viable. However, as the gangrenous changes in the cecum were irreversible, the cecum was resected. An end-to-end anastomosis was then made between the terminal ileum and the ascending colon.

#### Pathologist's Report

Extensive gangrenous changes were found throughout the cecal wall. No definite evidence of perforation was seen externally. However, an indurated area approximately 2 cm. in diameter was present in the area of the wall which had been found to be adherent to the pelvic floor at operation. Here again, no definite point of perforation was seen. However, the tissue was decidedly indurated and friable. When the cecum was opened, an ulcer crater was seen in the mucosal wall corresponding to the above described area on the outside of the cecal wall. The ulcer was approximately 1 cm. in diameter and 2 to 3 mm. deep. The floor of the ulcer was gray and the edges appeared to be undermined. Ad-

Submitted June 6, 1960.

jacent to the ulcer and throughout the mucosa of the cecum were multiple elevated grayish plaques with innumerable small ulcerations between them.

Microscopic sections through the cecal ulcer showed that the mucosal epithelium terminated rather acutely and was replaced by an ulcer floor consisting of collections of polymorphonuclear leukocytes. The cecal wall was almost completely destroyed. The inflammatory cells extended directly to the serosa. There was no muscularis or mucosal layer. A thin fibrinal purulent exudate came from the serosa. Sections throughout the remainder of the cecal wall showed similar small ulcerations of lesser severity. The pathological diagnosis was ulcerative colitis with gangrene of the cecum.


#### SUMMARY

A unique case of gangrene of the cecum owing to volvulus, in a patient who had ulcerative colitis, is presented. Bowel resection was necessary. The case draws attention anew to the necessity for careful observation for complications in patients with ulcerative colitis.



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## Tuberculosis of the Duodenum


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THE INCIDENCE OF TUBERCULOSIS in all forms has decreased since the advent of drugs specific for the disease, and tuberculosis of the duodenum is now more than ever a rarity. Matthews, Delaney, and Dragstedt[4] cited a report by Paerry and Shaw in 1894 that only 11 cases of duodenal tuberculosis were noted in a review of 17,652 autopsies that had been done at Guys Hospital over the previous 66-year period. They also cited Fenwick and Dodwell who in 1892 reported 500 cases of intestinal tuberculosis in 2,000 autopsies of patients with pulmonary tuberculosis, only 17 of the 500 having duodenal tuberculosis. Also quoted was Gossmann, who in 1913 noted five cases of duodenal tuberculosis in reviewing 2,360 autopsies of patients with tuberculosis. In the same communication in which they cited these reports, Matthews and co-workers added reports of 18 cases of duodenal tuberculosis, making 123 cases that had been reported in the literature up to 1932.

In 1931, Good (cited by Ostrum[6]) reported an incidence of 0.34 per cent of gastric tuberculosis in autopsies and gastric resections at the Mayo Clinic. Reeves[7] reported a case of tuberculosis of the duodenal bulb in 1931. In the Case Records of the Massachusetts General Hospital in 1942, Mallory[3] reported a tuberculous ulcer in the third portion of the duodenum, communicating with a retroperitoneal abscess cavity, associated with miliary tuberculosis. Migliaccio[5] in 1946 reported a case of tuberculosis of the third portion of the duodenum. Two years later Ostrum and Serber[6] reported two cases of duodenal tuberculosis, and in 1954 Anderson, Pontius and Witkowski[1] wrote of a case in the duodenal bulb.

#### REPORT OF A CASE

The patient, a 51-year-old Filipino, entered Wadsworth Hospital July 11, 1955, with chief complaints of epigastric pain for one year, with some relief on ingestion of food, and a 6-pound loss of weight in three months. There was one episode of a tarry stool. An upper gastrointestinal radiographic study done elsewhere one year before was said to show a duodenal ulcer. Upon physical examination, mild tenderness in the epigastrium was noted. No abnormalities were noted on examination of the blood and urine, and the serum amylase content was within normal limits.

Upper gastrointestinal radiographic studies were interpreted as showing an ulcerating and apparently infiltrating destructive mucosal lesion of the second portion of the duodenum (Figure 1). This was interpreted as suggestive for carcinoma of the duodenum with other possibilities being carcinoma of the ampulla of Vater or pancreas. A roentgenogram of the chest showed no abnormality.

At operation, July 19, an infiltrating lesion was observed in the descending duodenum. There were large nodes along the portal vein and hilus of the liver. Biopsy specimens were taken from thickened mucosal folds of the second portion of the duodenum and frozen sections were reported as showing possibly lymphosarcoma. A frozen section of a lymph node from the hilus of the liver showed caseous tuberculosis. Subtotal gastrectomy was done. However, pathological study of the surgical specimens (Figures 2 and 3) revealed tuberculous duodenitis with caseation of draining lymph nodes. A pulmonary embolus developed postoperatively and bilateral saphenous and femoral vein ligation was carried out. Pancreatitis also developed, the blood


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Submitted August 9, 1960.

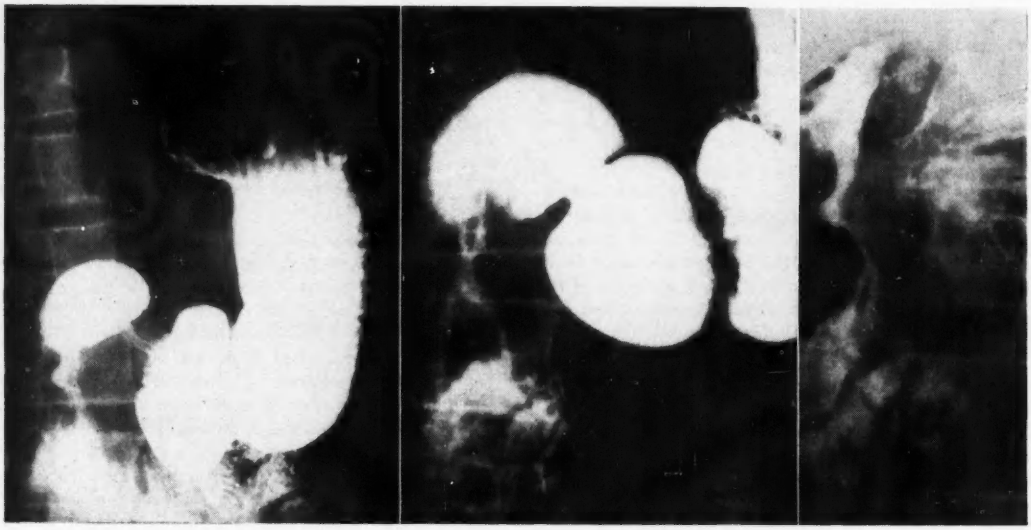

Figure 1.—*Left*, radiograph showing a long narrow infiltrating lesion of the second portion of the duodenum with ulceration. *Center*, spot film of second portion of duodenum, showing destroyed and distorted mucosa. *Right*, spot film of second portion of duodenum, showing ulceration and narrowing.

amylase rising to 18,000 units. The condition of the patient deteriorated rapidly and he died August 2.

At post mortem examination tuberculosis of the second portion of the duodenum was observed, with post-surgical changes and caseous tuberculous lymphadenitis of para-duodenal, gastro-hepatic and porta hepatis nodes. Generalized peritonitis, pulmonary embolism and focal hemorrhagic pancreatitis were also noted.

#### Symptoms and Physical Findings

The symptoms of duodenal tuberculosis are not specific. Sometimes there are no symptoms, sometimes symptoms are like those of duodenal ulceration, sometimes there are diffuse symptoms referrable to the gastrointestinal tract. There may be hematemesis, melena, loss of weight, pain and fever. Often the disease is associated with either active or arrested pulmonary tuberculosis. In some cases there is primary intestinal involvement.

The physical features usually are not specific or helpful in making a correct diagnosis. Occasionally non-specific epigastric tenderness may be present, and a mass may be palpated.

#### Pathology

Like intestinal tuberculosis, duodenal tuberculosis can be ulcerative or hyperplastic.

The ulcerative type is most common. Ulcers may be single or multiple. The tubercle bacilli reach the submucosal layer, producing caseation and ulceration into the lumen of the intestinal tract. The tuberculous ulcers may be scalloped, irregular and undermined or elevated. Tubercles are often seen like a string of pearls over the serosa and along the lymphatic chain. Adjacent lymph nodes are often involved. Fistulae, sinus tracts and perforations are common.

The hyperplastic type is characterized by a proliferative thickening of the bowel wall, producing a granulating, polypoid appearance of the mucosa which grossly may be indistinguishable from a neoplastic process. Ulceration is rare in this type.

Microscopically (Figures 2 and 3) characteristic tubercles have epithelioid and giant cells located in swollen follicles, and rarely tubercle bacilli. Miliary or sub-miliary tubercles, isolated or in groups, may be found in the mucosa, submucosa and muscularis, or in the margins of ulceration.

#### Radiographic Features

In roentgenographic studies the mucosa is usually irregular, involving a long segment, often with polypoid mucosal changes suggesting neoplasm, and may or may not be ulcerated. In some cases involving the duodenal bulb, the deformity is difficult to differentiate from a duodenal ulcer. A delay of passage of barium through the involved area of duodenum, with dilatation both proximal and distal to the lesion, is seen occasionally. Sinus tracts leading from the ulceration can be seen. As the lesion heals, scarring can occur and with it the development of obstruction by stenosis, angulation or retraction of the bowel by adhesive bands.

#### Operative Observations

In the case herein reported, and in several others previously reported, the lesion as seen at operation closely resembled neoplasm. There were thickened, nodular, proliferative, polypoid mucosal changes

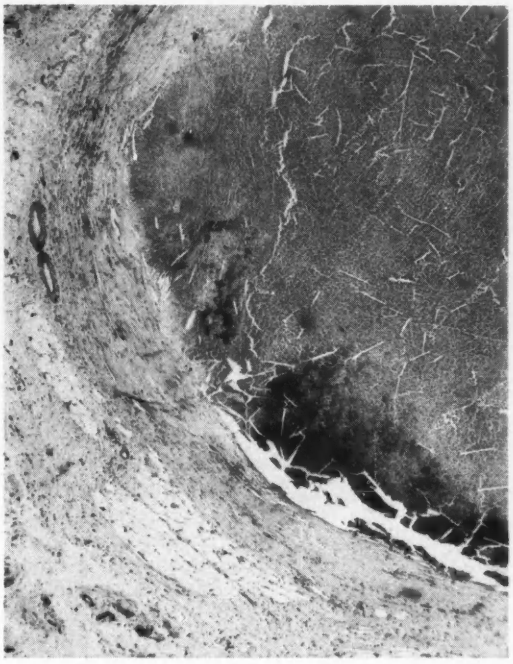

Figure 2.—Microscopic section (×75) showing caseous necrosis in an abdominal lymph node.

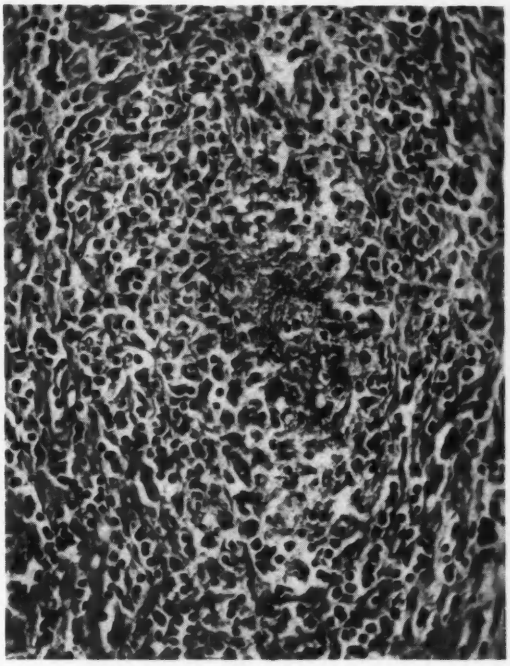

Figure 3.—Microscopic section (×150) showing a granuloma in the wall of the duodenum.

with associated enlarged lymph nodes. However, one may find sinus tracts with intraperitoneal or retroperitoneal abscesses, adhesions and tuberculous peritonitis.


## SUMMARY

A case report of ulcerative tuberculosis of the second portion of the duodenum with radiographic findings is given. Radiographically the lesion simulated both neoplasm and post bulbar ulceration of the duodenum. At operation it resembled a neoplasm.

Discussion of the symptoms, physical, radiographic, surgical and pathologic features of duodenal tuberculosis is presented.



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California MEDICINE

For information on preparation of manuscript, see advertising page 2

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# EDITORIAL

## C. P. S. - Blue Shield

CALIFORNIA PHYSICIANS' SERVICE was put together more than twenty years ago by a dedicated group of physicians who saw in the depression-bound 30's that the provision of medical services was becoming, in the minds of the people, a necessity which must be made to conform with family budgeting procedures.

In the twenty years since its founding C.P.S. has not only proved its ability to fulfill its original purpose but has demonstrated its ability and flexibility under a variety of circumstances which enable it to serve the people and the medical profession alike.

It has handled the home-town medical care problem of the Veterans' Administration for more than 13 years. It has handled the Medicare program for the dependents of service personnel since the inception of that program. It has served as fiscal agent for public welfare programs in the majority of the counties of the state. It has served as fiscal agent and underwriter for some of the local medical care programs embraced by foundations for medical care in several of the counties.

Most recently C.P.S. has emerged as the program of choice for 100,000 Californians who are federal employees and who chose the Blue Shield concept as their preferred method of providing medical and hospital care for themselves and their families.

In addition, C.P.S. has produced a medical care plan for the aged and has pioneered in a variety of empirical experiments in fields covering mental health, the aged, in-patient services and other areas.

Throughout this experience, C.P.S. has acted under the direct authority and control of the physicians of the state as represented in the House of Delegates of the California Medical Association. Its policies and directives emanate from the same body of representatives that sets policies for the 17,000 plus physicians who make up the state association.

To carry out these policies and directives, C.P.S. is managed by a Board of Trustees which includes both physicians and lay members. The physician members of the board are elected by the C.M.A. House of Delegates, with the exception of three who are named by the C.M.A. Council from its own membership. These Councilors also are elected by the House of Delegates. The lay members of the board are chosen from the business, public and religious interests of the people of California and are subject to confirmation by the House of Delegates. It is noteworthy that these lay members have consistently been executives who enjoy a high standing in their own fields of endeavor and who are willing to contribute their time and talents as a service to the public.

Today, after two decades of experience, C.P.S. would appear to be prepared to meet all challenges and to provide all services that are in the public interest, that recognize the needs and practices of its physician members and that are actuarially sound.

In spite of this apparent position, however, we find C.P.S. beset with a number of problems which a knowledgeable observer might have expected to see solved some years back.

Details need not be gone into here, but it is obvious that many of the problems of C.P.S. stem from its owners, the physicians. Some physicians have never approved of the interjection of a third party—even their own organization—into the normal physician-patient relationship. Some have never agreed that the service principle, under which the subscriber whose income is below an established level is provided professional services rather than dollars, is a good thing. And some physicians just don't want to be bothered. One measure of the general attitude of physicians, however, is that some 14,500 of them are C.P.S. members.

Many of these physicians, of course, interpret the service concept of medical care in terms of their own practices and activities. And many of them may be suspect of ignoring or failing to keep pace with

the drastic social changes which have occurred in recent years.

Whereas twenty years ago the employer paid an agreed wage and no more, employers today are averaging about 20 per cent of their payrolls as additional costs for fringe benefits. The fringe items almost invariably include some provision for medical and hospital care.

The giant strides made by all forms of voluntary health insurance underwriting under these conditions are apparent to all. They have regularly been used to combat the proposals of the more vocal do-gooders that all medical care should be made a function of government, paid for by taxation. Only the tremendous growth of the voluntary plans has stood in the way of the compulsory health insurance proposals made at both state and federal levels.

With the growing public interest and participation in the voluntary forms of health insurance has come the demand for certainty of coverage. Employers and employees alike are quick to protest when a service is provided and the physician sees fit to collect the insurance benefit and then add on an additional fee which becomes the personal responsibility of the employee. Some of this protest is reflected in the growth of the closed-panel systems which do not assess additional fees. Some of it shows up in the acceptance of indemnity programs by employee groups, accompanied by the formation of a panel of physicians in the area who agree to accept the indemnity allowances as full payment for services. In either event, it is obvious that the medical profession is being splintered.

It must be understood that only a physician, or a group of physicians such as in C.P.S., can sell medical services. The sale of these services by anyone else comes under the legal proscription against corporate medical practice. However, by combining an indemnity schedule of allowances with the agreement of individual physicians to accept these sums as payment in full, the promoters of some plans are able to drain away from the medical profession its inherent and exclusive right to market its own services as a professional skill.

Doubtless some of today's programs of this character are experimental and possibly visionary. They may seem to offer an immediate benefit despite long-range potentialities of danger.

Now would seem to be the time for all physicians to indulge in some clear thinking, to analyze both the good and the bad features of a variety of prepayment plans and to give credit where credit is due.

If this process is followed, C.P.S. will most certainly be credited with a number of assets which other plans cannot claim. It is the doctors' own plan, subject to their wish. It is a professional, rather than a commercial plan. It has the right, by nature and by law, to offer services rather than dollars. It has the public interest at heart. And, it represents physicians in a number of dealings with government where individual efforts would be lost. It operates locally, but in combination with its counterparts in other states it forms the nationwide Blue Shield and becomes a national power where such power is needed.

It is not our purpose here to decry other forms of voluntary health insurance. It is obvious to all that in so new a field of endeavor there must be competition and that such competition is effective in producing the greatest benefit for the greatest number of people.

It is our purpose to suggest that California Physicians' Service and the Blue Shield movement have within themselves a number of features which are designed for and by physicians. It is our further purpose to suggest that all California physicians give some serious thought to the future of medical practice under evolving social mechanisms and to consider which forms of prepayment medical care insurance offer the sound features which may be counted upon to be truly representative of the medical profession in the years to come.

Surely C.P.S. will stand high in such a list, if not at the pinnacle.

California MEDICAL ASSOCIATION

# NOTICES & REPORTS

## County Society Officers

### • A Meeting on *The Challenge to Private Practice*

A DAY-AND-A-HALF long conference of officers of county medical societies in California on various aspects of "The Challenge to the Private Practice of Medicine" will be held at the Biltmore Hotel, Los Angeles, February 11 and 12. Besides the component society representatives, all others interested are welcome to attend.

Sponsored by the California Medical Association, the conference will be addressed by outstanding speakers on medical economics, insurance plans, governmental medical care programs, medical care plans of trade unions, and legislative prospects. It will serve also for the indoctrination and instruction of the county society officers as to the relationship of the component societies to the functions and programs of the state association.

The program for the meeting, which is to last all day Saturday, February 11, and Sunday forenoon, follows:

SATURDAY

9:00—Registration.

9:30—Welcome; Format of Conference; Introduction of Chairman—Paul D. Foster, M.D., President.

PANEL NO. 1

**Trends in the Future of Private Practice**

Moderator: Malcolm C. Todd, M.D., Councilor, Third District

9:45—PROFESSOR WALTER J. MCNERNEY, Director, Program in Hospital Administration, School of Business Administration, University of Michigan, Ann Arbor, Michigan.

10:15—JOHN TOMAYKO, Director, Insurance, Pensions and Unemployment Benefits Department United Steelworkers of America, Pittsburgh, Penn.

10:45—CHARLES E. THOMPSON, M.D., Medical Consultant, Chicago Truck Drivers, Chauffeurs and Helpers Union of Chicago and Vicinity (Independent), Chicago, Illinois.

11:15—ROGER O. EGEBERG, M.D., Director, Los Angeles Department of Charities; Chairman, Governor's Committee on the Study of Medical Aid and Health.

11:45—Questions.

12:30—Luncheon. DONALD H. STUBBS, M.D., Chairman, Board of Directors, National Association of Blue Shield Plans, Washington, D. C.

PANEL NO. 2

**Trends in the Future of Voluntary Health Insurance**

2:30—Moderator: Donald C. Harrington, M.D., Chairman, Commission on Medical Services.

2:40—MRS. AGNES W. BREWSTER, Medical Economist, Division of Public Health Methods, Public Health Service, U. S. Department of Health, Education and Welfare, Washington, D. C.

3:10—JOSEPH F. FOLLMANN, JR., Director of Information and Research, Health Insurance Association of America, New York.

3:40—JAMES BRINDLE, Director, Social Security Department, United Auto Workers, Detroit, Michigan.

4:10—ODIN ANDERSON, Ph.D., Research Director, Health Information Foundation, New York.

4:40—Questions.

SUNDAY

PANEL NO. 3

**Government in Medical Care**

9:00—Moderator: Samuel R. Sherman, M.D., Chairman of the Council.

PAUL D. FOSTER, M.D. . . . . President
WARREN L. BOSTICK, M.D. . . . . President-Elect
JAMES C. DOYLE, M.D. . . . . Speaker
IVAN C. HERON, M.D. . . . . Vice-Speaker
SAMUEL R. SHERMAN, M.D. . . Chairman of the Council
RALPH C. TEALL, M.D. . . Vice-Chairman of the Council
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*The Social Movement—Its Implications to Private Practice:* THOMAS F. JOHNSON, Director of Research, American Enterprise Association, Washington, D. C.

*Canadian Experiences:* M. R. MACCHARLES, M.D., Vice-President, Trans-Canada Medical Plans, Winnipeg, Manitoba.

*The California Picture:* DAN O. KILROY, M.D., Chairman, Committee on Legislation.

*The National Picture:* C. JOSEPH STETLER, Director, Division of Socio-Economics, AMA, Chicago, Illinois.

Questions.

11:45—1961 House of Delegates: JAMES C. DOYLE, M.D., Speaker of the House.

1961 Secretaries' Manuals: JOHN HUNTON, Executive Secretary.

12:30—Adjournment.

## Joint News Release By CMA and COA

*The following news story was sent jointly by the California Medical Association and the California Osteopathic Association to newspapers in California for release December 15, 1960.*

Further progress in the long series of negotiations between the leaders of the California Medical Association and the California Osteopathic Association based upon mutual desires for the over-all improvement in medical care for all Californians through the anticipated unification of the two professions was announced here today.

Only broad principles, according to the joint announcement by the two associations, have been agreed upon thus far. The details of any final agreement between the members of the two professions, it was pointed out, will have to be approved by the respective House of Delegates meetings at their 1961 sessions.

Both presidents, Doctor Paul D. Foster of CMA and Doctor Dorothy J. Marsh of COA, were in total agreement that the contemplated unification would:

1. Lead to the increase in the total number of qualified physicians and surgeons to serve the state's growing population;

2. Provide additional postgraduate opportunities for the practicing physicians of the state;

3. Unite the two groups with a renewed determination to preserve the freedom of the doctors to provide the best possible patient care.

The Board of Osteopathic Examiners was established by the California voters in 1922 as a part of the "Osteopathic Act." The Board, by law, issues licenses to osteopathic physicians and surgeons which grant them the same privileges as medical doctors who have been licensed by the State Board of Medical Examiners.

Examinations for both groups are identical.

The standards set by the California Osteopathic Board, it is generally agreed, are higher than any other state where this type of license is granted.

## In Memoriam

BAILEY, OLIVER LADBROOK. Died in San Francisco, November 19, 1960, aged 59. Graduate of the University of Toronto Faculty of Medicine, Ontario, Canada, 1934. Licensed in California in 1947. Doctor Bailey was a member of the San Francisco Medical Society.

✤

BAKER, BLANCHE M. Died in San Francisco, December 11, 1960, aged 54. Graduate of Ohio State University College of Medicine, Columbus, 1939. Licensed in California in 1946. Doctor Baker was a member of the San Francisco Medical Society.

✤

DAVIS JAMES VALENTINE. Died in Glendale of heart disease, December 11, 1960, aged 54. Graduate of Columbia University College of Physicians and Surgeons, New York, 1936. Licensed in California in 1946. Doctor Davis was a member of the Los Angeles County Medical Association.

✤

GATES, MORRIS J. Died in Santa Cruz, November 17, 1960, aged 90. Graduate of the Kentucky School of Medicine, Louisville, 1894. Licensed in California in 1894. Doctor Gates was a member of the Santa Cruz County Medical Society, a life member of the California Medical Association, and a member of the American Medical Association.

GREENHALGH, MARLIN DOUGLAS. Died in an airplane crash, November 20, 1960, aged 48. Graduate of the University of Utah College of Medicine, Salt Lake City, 1950. Licensed in California in 1950. Doctor Greenhalgh was a member of the Sonoma County Medical Society.

✤

KELLETT, MACK A. JR. Died in Northridge, November 10, 1960, aged 40. Graduate of the University of Arkansas School of Medicine, Little Rock, 1944. Licensed in California in 1950. Doctor Kellett was a member of the Los Angeles County Medical Association.

✤

POLITO, SANTO SALVATORE. Died in Burbank, November 19, 1960, aged 49. Graduate of the University of Buffalo School of Medicine, New York, 1936. Licensed in California in 1944. Doctor Polito was a member of the Los Angeles County Medical Association.

✤

REEVES, JOHN MURPHY. Died December 10, 1960, aged 70. Graduate of Vanderbilt University School of Medicine, Nashville, Tennessee, 1921. Licensed in California in 1923. Doctor Reeves was a member of the Alameda-Contra Costa Medical Association.

# 1961

*For complete program see March issue*

# *Annual Session*

**AMBASSADOR HOTEL • LOS ANGELES**

**APRIL 30 to MAY 3**

- Five Outstanding Guest Speakers
- General Scientific Meetings
- Specialty Scientific Meetings
- Postgraduate Courses
- Medical Motion Picture Symposia
- Technical Exhibits • Scientific Exhibits
- Presidents' Dinner Dance
  Sunday, April 30 • Cocoanut Grove
- House of Delegates
  Opening Session Saturday, 2:00 p.m., April 29
  Sunday, April 30, Tuesday Afternoon, May 2, and Wednesday, May 3
- Registration Daily
  8:30 a.m. to 5:00 p.m. . . . No Registration Fee

PLEASE MAKE HOTEL ROOM RESERVATIONS ONLY THROUGH C.M.A. OFFICE IN SAN FRANCISCO. USE RESERVATION REQUEST FORM ON PAGE 47.

# C.M.A. ANNUAL MEETING—1961 SCIENTIFIC SESSIONS

| LOCATION | SUNDAY APRIL 30 | | MONDAY MAY 1 | | TUESDAY MAY 2 | | WEDNESDAY MAY 3 | |
|---|---|---|---|---|---|---|---|---|
| **AMBASSADOR HOTEL** (Lobby Floor) | A.M. | P.M. | A.M. | P.M. | A.M. | P.M. | A.M. | P.M. |
| Embassy Room | **9:30 a.m.†** **House of Delegates** †See note below for Opening Session | **2 p.m.** General Meeting Inflammatory Diseases of the Colon | **9 a.m.** General Meeting Diagnostic Methods of Gastroenterology | | **9 a.m.** General Meeting Five Guests Five Topics | **House of Delegates** | **House of Delegates** | |
| West Venetian Room | **9 a.m.** Internal Medicine | **2 p.m.** Film Symposium | **9 a.m.** Film Symposium | **2 p.m. & 8 p.m.** Film Symposia | **9 a.m.** Film Symposium | **2 p.m. & 8 p.m.** Film Symposia | **9 a.m.** Film Symposium | **2 p.m.** Film Symposium |
| East Venetian Room | **9 a.m.** Pathology | | | | | **2 p.m.** Industrial Medicine Physical Medicine General Practice | | **2 p.m.** Public Health |
| Regency Room | **9 a.m.** Obstetrics and Gynecology | **2 p.m.** Eye Anesthesiology | | **2 p.m.** Anesthesiology | | **2 p.m.** Obstetrics and Gynecology Anesthesiology | **9 a.m.** Neurology | **2 p.m.** Psychiatry |
| Grove Lounge | **9 a.m.** Dermatology | **2 p.m.** Ear, Nose, Throat | | | | | **9 a.m.** Urology | **2 p.m.** Urology |
| Lido Room | **9 a.m.** Radiology | **2 p.m.** Radiology | | | **9 a.m.** Physical Medicine | | | **2 p.m.** Internal Medicine |
| Colonial Room | **9 a.m.** General Surgery | | | | | **2 p.m.** Disaster Medical Care | | |
| Rose Room | **9 a.m.** Orthopedics | | | | | | | |
| (Casino Floor) Oval Room A | **9 a.m.** Allergy | **12 noon** Allergy Luncheon and Business Meeting | | | | | | **2 p.m.** Pediatrics |
| **WHITE MEMORIAL HOSPITAL** | **9 a.m.*** **(Bus at 8 a.m.*)** Postgraduate Course C.M.E. | | | **2 p.m.*** **(Bus at 1 p.m.*)** Postgraduate Course C.M.E. | | **2 p.m.*** **(Bus at 1 p.m.*)** Postgraduate Course C.M.E. | | |
| **L. A. COUNTY HOSPITAL** (Starts Saturday) | **9 a.m.*** **(Bus at 8 a.m.*)** Postgraduate Courses U.S.C. | **2 p.m.** **(No bus)** P.G. Course—Cardiac Resuscitation—U.S.C. | | | | | | |

*Buses will leave Ambassador Hotel, Wilshire Entrance.
U.S.C. and U.C.L.A. Courses will start on Saturday.
**†Opening meeting, House of Delegates, 2:00 p.m., Saturday, April 29.**

TECHNICAL EXHIBITS—Sunset Room, Ballroom and Boulevard Room, Casino Floor.
SCIENTIFIC EXHIBITS—Lido Patio, Lobby Floor.
COUNCIL OF THE C.M.A. MEETS DAILY AT 7:30 A.M. IN THE FRENCHETTE ROOM.

# 4 POSTGRADUATE COURSES

## C.M.A. ANNUAL SESSION • *April 29–May 3, 1961* • *Los Angeles*

THE CALIFORNIA MEDICAL ASSOCIATION *in cooperation with the Medical Schools of* UNIVERSITY OF SOUTHERN CALIFORNIA *and* COLLEGE OF MEDICAL EVANGELISTS, *will present four Postgraduate Courses at the time of the Annual Session in April. These courses will be clinically oriented and will include case presentations.*

*Choose the course which most interests you, follow the course, and the 1961 session will send you back to your practice stimulated and refreshed.*

*Look for the program giving complete details which will arrive in your office in March.*

**• By COLLEGE OF MEDICAL EVANGELISTS**

### Clinical Neurology—9 hours

*Time:* Sunday, April 30, 9:00 a.m.† to 12:00 noon; Monday and Tuesday, May 1 and 2, 2:00 p.m.† to 5:00 p.m.
*Place:* White Memorial Hospital. *Fee:* $25.00

**• By UNIVERSITY OF SOUTHERN CALIFORNIA**

### Uses and Limitations of Laboratory Tests—9 hours

*Time:* Saturday, April 29, 9:00 a.m.† to 12:00 noon; 2:00 p.m.‡ to 5:00 p.m.; Sunday, April 30, 9:00 a.m.† to 12:00 noon.
*Place:* Los Angeles County Hospital. *Fee:* $25.00

### Office Gynecology—9 hours

*Time:* Saturday, April 29, 9:00 a.m.† to 12:00 noon; 2:00 p.m.‡ to 5:00 p.m.; Sunday, April 30, 9:00 a.m.† to 12:00 noon.
*Place:* Los Angeles County Hospital. *Fee:* $25.00

### Cardiac Resuscitation—2 hours

SECTION I*—Saturday, April 29, 2:00 p.m.‡ to 4:00 p.m.
SECTION II*—Sunday, April 30, 2:00 p.m.‡ to 4:00 p.m.
*Place:* Los Angeles County Hospital. *Fee:* $30.00

*Each Section is all-inclusive. Each Section is limited to 15 registrants from areas other than San Francisco or Los Angeles.

† ‡ Bus transportation is provided to reach the courses at the times marked with a dagger (†), but not for the times marked with a double dagger (‡). The buses will leave the Ambassador Hotel, Wilshire entrance, at 8:00 a.m., for the courses that begin in the mornings, and at 1:00 p.m. for the courses that begin in the afternoons.

**APPLICATION FOR ENROLLMENT**

*Mail to:* POSTGRADUATE ACTIVITIES, CALIFORNIA MEDICAL ASSOCIATION
2975 Wilshire Boulevard, Los Angeles 5, California

With check or money order in the amount of $_____ made payable to *California Medical Association*

Name _____

Address _____

I am in General Practice _____ I limit my practice to _____

Medical School Attended _____ Year of Graduation _____

*Please enroll me in the course indicated by ✓.*

- ☐ 1. **Clinical Neurology** (9-hour course, Sunday morning; Monday and Tuesday afternoons)—$25.00
- ☐ 2. **Uses and Limitations of Laboratory Tests** (9-hour course, Saturday, all day; Sunday morning)—$25.00
- ☐ 3. **Office Gynecology** (9-hour course, Saturday, all day; Sunday morning)—$25.00
- ☐ 4a. **Cardiac Resuscitation—Section I**—Saturday afternoon—$30.00
- ☐ 4b. **Cardiac Resuscitation—Section II**—Sunday afternoon—$30.00

# APPLICATION FOR HOUSING ACCOMMODATIONS

FOR YOUR CONVENIENCE in making hotel reservations for the coming meeting of the **California Medical Association,** April 30*-May 3, 1961, Los Angeles, hotels and their rates are at the right. Use the form at the bottom of this page, indicating your first and second choice. Because of the limited number of single rooms available, your chance of securing accommodations of your choice will be better if your request calls for rooms to be occupied by two or more persons. **All requests for reservations must give definite date and hour of arrival as well as definite date and approximate hour of departure; also names and addresses of all occupants of hotel rooms must be included.**

# Ninetieth Annual Session
## CALIFORNIA MEDICAL ASSOCIATION
## Los Angeles, California
## APRIL 30*-MAY 3, 1961

## HOTEL ROOM RATES†

| | Single | Twin Beds | Suites |
|---|---|---|---|
| **AMBASSADOR HOTEL** | | | |
| 3400 Wilshire Boulevard | | | |
| Main Building | 14.00-24.00 | 18.00-28.00 | 40.00-58.00 |
| Garden Studios | 22.00-34.00 | 24.00-36.00 | 54.00-66.00 |
| **CHAPMAN PARK HOTEL** | | | |
| 3405 Wilshire Boulevard | 10.00-11.00 | 14.00-16.00 | 20.00-28.00 |
| Bungalows | | 18.00 | 25.00-28.00 |
| **THE GAYLORD HOTEL** | | | |
| 3355 Wilshire Boulevard | 9.00-10.00 | 12.00-15.00 | Single: 25.00<br>Double: 35.00 |
| **HOTEL CHANCELLOR** | | | |
| 3191 West Seventh Street | 8.00-10.00 | 12.00 | |
| **SHERATON-WEST** (formerly Sheraton-Town House) | | | |
| 2961 Wilshire Boulevard | 12.50-20.00 | 17.50 | 34.00 |

**ALL RESERVATIONS MUST BE RECEIVED BEFORE: APRIL 1, 1961**

***April 29: House of Delegates will start with afternoon meeting Saturday, April 29.**

†The above quoted rates are existing rates but are subject to any change which may be made in the future.

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CALIFORNIA MEDICAL ASSOCIATION—Dept. 74
693 Sutter Street
San Francisco 2, California

Please reserve the following accommodations for the 90th Annual Session of the California Medical Association, in Los Angeles April 30-May 3, 1961. (House of Delegates members: First meeting of House begins Saturday afternoon, April 29.)

Single Room $..... Twin-Bedded Room $.....

Small Suite $..... Large Suite $..... Other Type of Room $.....

First Choice Hotel..... Second Choice Hotel.....

ARRIVING AT HOTEL (date):..... Hour:..... A.M. P.M. } Hotel reservations will be held until

Leaving (date) ..... Hour:..... A.M. P.M. } 6:00 p.m., unless otherwise notified

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Therefore, please include the names of both persons for each twin-bedded room requested. Names and addresses of all persons for whom you are requesting reservations and who will occupy the rooms asked for:

.....

.....

.....

.....

Individual Requesting Reservations—Please print or type Officer?..... Delegate?..... Alternate?.....

Name..... County.....

Address..... City and State.....

11th ANNUAL

# POSTGRADUATE INSTITUTE

## SOUTHERN COUNTIES

Presented by U.S.C. School of Medicine, Phil R. Manning, M.D., Associate Dean Postgraduate Division, and California Medical Association Committee on Postgraduate Activities.

Guest Speaker: Jerome W. Conn, M.D., Professor of Medicine, University of Michigan.

*El Mirador Hotel, Palm Springs*

**February 2 and 3, 1961**

## PROGRAM

### THURSDAY, FEBRUARY 2

*Morning Meetings*

9:15-10:15—**Primary Aldosteronism and Hypertensive Disease**—Jerome W. Conn, M.D.

10:30-11:00—**The Office Approach to Low Back Pain**—Lorin L. Stephens, M.D.

11:00-11:30—**Further Advances in the Tranquilizers**—John L. Webb, Ph.D.

11:30-12:30—**Rheumatic Fever**—George C. Griffith, M.D.

ELECTIVES

10:30-12:30—**Workshop in Jaundice**—Telfer B. Reynolds, M.D.

10:30-12:30—**Pitfalls in the Diagnosis of Secondary Hypertension and Use of the Ophthalmoscope in Evaluation of Hypertension**—Phil R. Manning, M.D.; Richard L. Kasper, M.D.

*Afternoon Meetings*

2:00-3:20—**Panel on Gallbladder Diseases**—Clarence J. Berne, M.D., Bernard J. Haverback, M.D., Telfer B. Reynolds, M.D.

3:30-4:00—**Difficulties in Diagnosis and Treatment in the Otitis Media**—Irwin Harris, M.D.

4:00-4:30—**The Practical Aspects of Newer Advances in Endocrinology**—Don H. Nelson, M.D.

4:30-5:00—**Management of Breech Presentation**—Gail V. Anderson, M.D.

ELECTIVES

2:00-5:00—**Motion Picture Symposium on Surgery**—Raymond F. Tatro, M.D., Moderator.

3:30-5:00—**Workshop in Cardiovascular Diagnosis**—George C. Griffith, M.D.

7:00-8:00—**"No Host" Cocktail Party.**

8:00—**Informal Dinner and Dancing.**

### FRIDAY, FEBRUARY 3

*Morning Meetings*

9:15-10:15—**Helpful Hints in the Recognition and Treatment of Various Types of Spontaneous Hypoglycemia**—Jerome W. Conn, M.D.

10:30-11:00—**The Sore Shoulder and Neck**—Lorin L. Stephens, M.D.

11:00-11:30—**Use of Amine Oxidase Inhibitors**—John L. Webb, Ph.D.

11:30-12:30—**Selection of Patients for Cardiac Surgery**—Leonard H. Schwartz, M.D.

ELECTIVES

10:30-12:30—**Workshop in the Use of Antibiotics**—Phil R. Manning, M.D.

10:30-12:30—**Discussion of Problem Obstetrical Cases**—Gail V. Anderson, M.D.

*Afternoon Meetings*

2:00-3:20—**Panel on Duodenal Ulcer**—Clarence J. Berne, M.D., Bernard J. Haverback, M.D., Telfer B. Reynolds, M.D.

3:30-4:00—**Diagnosis and Treatment of Adrenal Disorders**—Don H. Nelson, M.D.

4:00-4:30—**ENT Problems in Allergic Children**—Irwin Harris, M.D.

4:30-5:00—**Management of Bleeding Problems During Prenatal Period**—Gail V. Anderson, M.D.

ELECTIVES

3:30-5:00—**Printed Case Discussions**—Telfer B. Reynolds, M.D.

3:30-5:00—**Workshop in Medical Ophthalmology**—Richard L. Kasper, M.D.

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**HOST:** San Bernardino County Medical Society . . . **REGIONAL CHAIRMAN:** Raymond F. Tatro, M.D., 491 Fifth Street, San Bernardino. **INSTITUTE FEE:** $15.00. For additional information contact Postgraduate Activities office, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5. **All California Medical Association members and their families are cordially invited to attend.**

# PUBLIC HEALTH REPORT

**MALCOLM H. MERRILL, *M.D., M.P.H.***
***Director, State Department of Public Health***

THERE WERE 121,523 cases of notifiable infectious diseases reported through November, compared with 116,401 cases in 1959. Among the most frequently occurring infectious diseases, there was a significant increase in streptococcal infections, which topped the 1960 list with 28,800 reported cases, through November, compared with 22,361 for all of 1959. Cases of venereal diseases, mumps and viral hepatitis also increased in number. There was a substantial decrease in cases of measles and pertussis.

The current trend as regards venereal disease is unfavorable. There have been substantial increases in reported cases of both infectious syphilis and gonorrhea.

Tuberculosis ranks sixth among notifiable diseases in the number of new cases. It is the cause of half the deaths due to notifiable diseases. Preliminary figures indicate that the number of new cases in 1960 will decline less than at any time in the past ten years. Similarly, the deaths from this disease are expected to show a very small decline. The decline may be due to stabilized disease rates in older persons and, in some areas of the state, to improved reporting.

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More than 12,750 radiation installations have been registered with the department. Current estimates are that this represents at least 75 per cent of all radiation source possessors in the state.

About 45 per cent of the registrants thus far are dentists, and almost 50 per cent are physicians, representatives of hospitals and x-ray laboratories, veterinarians, chiropractors and podiatrists. Most of the remaining registrations are for industrial, governmental, or educational facilities.

X-ray rachines are the most frequently reported radiation source. These include dental x-ray units, medical radiographic, fluoroscopic, and therapeutic equipment; industrial x-ray apparatus, with such diversified uses as the measurement of the level of beer in cans, the detection of flaws in metal casings, x-ray units for the inspection of prison visitors, and x-ray diffraction units for research and analysis of materials.

Aside from the knowledge gained through registration of who has radiation sources and why, important data concerning the safety practices of source possessors are becoming available. Registrants are reporting information concerning radiation safety surveys, or the lack of them, and the use of shielding, beam limitation devices, and filters to reduce or eliminate unnecessary exposure.

It is expected this information will be helpful in pointing out areas in which practices and equipment may be improved, where educational emphasis may be placed, and where controls or inspection are necessary.

The cooperation of registrants and of others associated with the program has been deeply appreciated. The information collected with their assistance should help make radiation a less hazardous, more useful tool to everyone.

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Three new projects in local health departments were approved recently by the department, including one for the development and evaluation of a family treatment program for the control of delinquency in Imperial County.

The Los Angeles City Health Department proposes to carry out a new program for the early detection of cervical cancer among patients receiving prenatal care in health department clinics. This program will be carried out in cooperation with the cytology laboratory of the Los Angeles County Hospital, and includes training of staff members in the necessary techniques.

That department also proposes to conduct a training program for selected public health administrators to improve their skills of handling the human problems of organizational administration and supervision.

# WOMAN'S AUXILIARY
## TO THE CALIFORNIA MEDICAL ASSOCIATION

### Auxiliaries' Christmas Activities

I SHOULD LIKE to use this page to report some of the very fine Christmas projects which the auxiliaries carried out in their various communities. Generally speaking, these activities fall into two major categories: (1) those associated with community services, and (2) those of a philanthropic nature.

The Sonoma County Auxiliary held a family Christmas party which included the doctors, their wives and children. The program, which was put on by the children, consisted of a choir, instrumental group, ballet and skits, and was under the jurisdiction of auxiliary members. Christmas gifts were donated by the members to needy children in foster homes. These gifts consisted of sweaters, skirts, wristwatches, and toys. The list of recipients was obtained from the county probation office.

In Los Angeles, the members conducted a tea to which they brought children's clothing, plus one dollar each, for gifts to be given through the Juvenile Bureau in Long Beach. Some of the auxiliary members accompanied the Future Nurses Club and decorated the wards on the U.S.S. Haven and sang Christmas carols. Other auxiliary members made dolls and toys that were given to the children's wards in the hospital. Also, Christmas favors were presented to patients at the Christian Home of Aged.

In Santa Barbara, at their annual Christmas Tea honoring new and prospective members, members brought gifts for the Council of Christmas Cheer. This year they were requested to bring a new gift, unwrapped, for a child up to age 15. The Council of Christmas Cheer coordinated the giving of some measure of Christmas cheer in the form of food, clothing, toys or cash for needy families or individuals referred by social agencies and schools. The Council maintains a central file to prevent duplication or omission of needy families or individuals referred to the Council. The Hospitality Committee of the auxiliary also made unusual decorations to sell at this tea. The money derived from this sale was to be used for some special need.

The San Diego Auxiliary selected a private home for the aged—the Golden Rule Rest Home—to be the recipient of their contributions. The supervisor of the home was asked to find out the particular needs of each patient. These consisted of nightgowns, bed jackets, bed slippers, etc. The auxiliary members made the necessary purchases and Christmas-wrapped them. Additional items of a non-specialized nature were brought to the December meeting by the members. Auxiliary members gave a party at the Home, replete with singers, refreshments and gifts. This excellent community project served a dual purpose: one, a community need, the other, attention to our aging population.

In Stanislaus County, each couple brought an unwrapped toy or cash gift to their annual Christmas dinner dance. The money and toys were turned over to their Salvation Army and distributed through those facilities.

The Mendocino-Lake Auxiliary had two projects this Christmas season. They bought and decorated a tree for the Geriatric Ward at the county hospital, and they decorated a tree for their newly established Point Two School (for severely mentally retarded children) and purchased a gift for each child.

In Solano County it has been customary for the auxiliary to adopt a ward at the county hospital. This year they "adopted" the Women's Ward. They donated individual gifts to each patient. Their "big" donation was painting, and installing window shades for the ward. Solano, like so many of our auxiliaries, also assisted in the preparation of the TB Christmas Seals and their mailing.

Riverside Auxiliary supplied toys, used or new, to the Community Settlement House. The members prepared baskets for needy families whose names were obtained from the welfare agencies.

Orange County's contribution has been a most active and diligent work activity in the TB Christmas Seal program. This involves the fulltime participation of six members every morning of the week (five mornings) for a period of two months.

In San Bernardino, the auxiliary members worked with Santa Claus, Inc., a local organization which helps needy families at Christmas time.

MRS. SAMUEL GENDEL,
*President, Woman's Auxiliary to the California Medical Association*

# NEWS & NOTES

## NATIONAL · STATE · COUNTY

### ALAMEDA

**A research grant** to total over $87,000 for a four-year period has been awarded to Dr. Edward A. Free at Children's Hospital of the East Bay **for a study of ascites.** The award was made by the National Institutes of Health of the U. S. Department of Health, Education, and Welfare.

Dr. Free is a research assistant at the Bruce Lyon Memorial Research Laboratory at Children's Hospital of the East Bay. He is also the resident in thoracic and cardiovascular surgery.

### LOS ANGELES

**Dr. Edward H. Crane** has been installed as president of the Los Angeles County Medical Association for 1961. Dr. Joseph P. O'Connor, who was the 1960 president, was elected a trustee. Dr. Roger Barnes was elected vice-president, and Dr. Ian Macdonald was reelected secretary-treasurer.

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The 29th annual **Alumni Postgraduate Medical Convention of the College of Medical Evangelists** will be held March 12-16, at the Ambassador Hotel and White Memorial Hospital, Los Angeles.

Refresher courses will be given in internal medicine, surgery, obstetrics and gynecology, and pediatrics. They are approved for **credit by the American Academy of General Practice.** Courses will be in session from 9:00 a.m. to 5:00 p.m. at White Memorial Hospital March 12 and 13.

On March 14, 15 and 16 a series of scientific lectures and panel discussions will be presented at the Ambassador Hotel. A total of 18 national and local medical authorities will participate in these presentations.

Further information may be obtained from the Office of Public Information, College of Medical Evangelists, 1720 Brooklyn Avenue, Los Angeles 33.

### SAN FRANCISCO

**Dr. Malcolm S. M. Watts** was installed as president of the San Francisco Medical Society at a meeting in December, succeeding Dr. Roberta Fenlon, and Dr. Eugene M. Webb was elected president-elect. Dr. Edgar Wayburn was elected secretary, succeeding Dr. John B. Schaupp, and Dr. Albert G. Clark was elected treasurer to succeed Dr. W. Dayton Clark. Dr. Helen B. Weyrauch was named editor of the *Bulletin*.

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At the annual meeting of the San Francisco chapter of the **Pan American Medical Association** the following officers were elected for 1961: President, Dr. J. Randolph Sharpsteen; first vice-president, Dr. Ray C. Atkinson; second vice-president, Dr. John W. Ashford; treasurer, Dr. Ivan A. May; secretary, Dr. Henry W. Weyrauch; chairman of regional administrators, Dr. Charles Pierre Mathé.

Drs. Paul G. Fuerstner, Saul T. Hersh and James Ownby, Jr., were elected to vacancies on the board of regional administrators.

**Dr. John J. Osborn** of Presbyterian Medical Center has been awarded a grant of $6,900 by the San Francisco Heart Association to support research on postoperative care of children who have had cardiac operations.

### SAN MATEO

**Dr. Frank J. Novak,** Menlo Park, was installed as president of the San Mateo County Medical Society for 1961 at the annual meeting in December, succeeding Dr. Robert O. Holmes. Dr. Frank T. Saidy, San Mateo, was elected president-elect, and Dr. John W. Findley, secretary.

### SAN BERNARDINO

**Dr. Charles P. Sprague,** San Bernardino, became the new president of the San Bernardino Medical Society at the annual meeting of the society in December. He succeeded Dr. Elmer O. Carlson of Ontario. Dr. William L. Cover of San Bernardino was elected president-elect, Dr. Merlin A. Hendrickson of Rialto, vice-president, and Dr. George W. Smith of San Bernardino, secretary-treasurer.

### VENTURA

**Dr. Charles Smolt,** Ventura, was installed as the 1961 president of the Ventura County Medical Society at a meeting of the society in December. Dr. Smolt succeeded Dr. John P. Pearson of Oxnard. Dr. Frank K. Helbling was reelected secretary.

### YOLO

**Dr. Herbert Bauer,** health officer of Yolo County, was elected president of the **California Conference of Local Health Officers** at the organization's annual meeting in November. He succeeded Dr. Everett M. Stone, Riverside County Health Officer. Dr. Dwight Bissell, health officer for the City of San Jose, was elected vice-president, and Dr. Robert S. Westphal, Stanislaus County, was elected secretary. The Conference is made up of all the local health officers in California.

### GENERAL

The Fourteenth annual meeting of the **Western Society for Clinical Research** will be held January 26-28 at the Golden Bough Theater, Carmel.

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The state of California has been the principal beneficiary in the **allocation of March of Dimes funds** raised in the state in the past 23 years, according to a financial summary prepared by The National Foundation.

More than 92 cents of every dollar from California's March of Dimes has been put to use in aiding the state's disease victims and in research and education projects conducted by California institutions, the report said. Of the remaining 8 per cent accruing to the national headquarters, a large portion also has been returned to California in shipments of poliomyelitis vaccine and gamma globulin and in other nationwide services conducted by the National Foundation.

The summary covers the period since the first March of Dimes was held in January, 1938, and compares the net total of funds raised in the state with amounts made available to California through September 30, 1960.

"In this period, California's 61 chapters of the March of Dimes organization raised a net total of $46,371,780.74 at an

**average fund-raising cost of 10.5 per cent,"** according to the report, and "of this amount, $34,064,052.96 has been available to the county chapters in carrying out their extensive patient aid programs, including advances of $12,660,830.87 from the national office to meet local emergency situations.

"In addition," the report continued, "247 grants totaling $8,710,322.56 have been made in **support of research and professional education** projects at California institutions. Principal recipients of these grants have been: The University of California, Berkeley, $2,135,197.91; Stanford University, $1,692,462.50; Rancho Los Amigos, $1,659,769.68; California Institute of Technology, $1,342,968.05; University of Southern California, $774,281.09; Alameda County Board of Supervisors, $332,173.69; California State Department of Public Health, $262,570.21; Orthopaedic Hospital, Los Angeles, $123,729.74; University of California, Los Angeles, $100,323.79 and Children's Hospital, Los Angeles, $38,663.93. Fairmont Hospital has been supported to the extent of $666,838.02 through the grants to Stanford University and the Alameda County Board of Supervisors.

## POSTGRADUATE EDUCATION NOTICES

THIS BULLETIN of the dates of postgraduate education programs and the meetings of various medical organizations in California is supplied by the Committee on Postgraduate Activities of the California Medical Association. In order that they may be listed here, please send communications relating to your future medical or surgical programs to Postgraduate Activities, California Medical Association, 2975 Wilshire Boulevard, Los Angeles 5.

### UNIVERSITY OF CALIFORNIA AT LOS ANGELES

**Clinical Traineeships — Anesthesia, Dermatology and Pediatric Cardiology.** Dates by arrangement. Minimum period—two weeks. Fee: Two weeks, $150.00; four weeks, $250.00.

**Below-Knee Prosthetics.** Monday through Friday, January 23 through 27. Enrollment limited to 20. Fee: $125.00.

**Cleft Palate Rehabilitation.** Friday through Sunday, January 27 through 29. Eighteen hours. Open to dentists, physicians and others interested. Fee: Full course: $75.00; two days: $65.00; One day: $10.00.

**Management of Fractures and Dislocation, and Application of Casts, Splints and Bandages.** Thursday through Saturday, February 23-25. Eighteen hours. Fee: Lecture and Lab $70.00; Lecture only $40.00.

**Use of Indirect Ophthalmoscopy in Retinal Detachment Surgery.** Thursday through Saturday, March 2-4. Eighteen hours.*

**Psychiatry in Medicine.** Friday and Saturday, March 10 and 11. Twelve hours. Fee: $2.50.

**Management of Pain by Therapeutic Nerve Blocks** —Harbor Hospital. Friday through Sunday, April 7 through 9. Eighteen hours. Fee: $55.00 (includes two luncheons).

*Fee to be announced.
†Hours and fees to be announced.

**Israel—Clinical Postgraduate Program** (sessions to be held in Jerusalem and Tel Aviv). April 20 through 28. Thirty-two hours. Fee: $200.00.

**Management of Trauma**—Harbor Hospital. Friday and Saturday, May 19 and 20. Nine hours.*

**Gerontology.** Friday and Saturday, May 19 and 20. Twelve hours.*

**Common Emergencies in Clinical Practice.** Friday and Saturday, May 26 and 27. Twelve hours. Fee: $40.00.

**Dermatology in Clinical Practice.** Tuesday, July 11. Six hours. Fee: $20.00.

**Advanced Seminars in Dermatology (for Dermatologists).** Wednesday through Sunday, July 12 through 16. University Conference Center, Lake Arrowhead. Fourteen and one-half hours. Fee: $150.00 (includes room and meals).

**Infertility.** Friday and Saturday, July 14 and 15.†

**Advanced Seminar on Infertility.** Sunday through Wednesday, July 16 through 19. University Conference Center, Lake Arrowhead.†

**General Pediatrics.** Wednesday through Sunday, August 2 through 6. University Conference Center, Lake Arrowhead. Sixteen hours. Fee: $150.00 (includes room and meals).

**Endocrinology.** Friday and Saturday, August 4 and 5.†

For information on courses for physicians or ancillary personnel *contact:* Thomas H. Sternberg, M.D., assistant dean for Continuing Medical Education, U.C.L.A. Medical Center, Los Angeles 24. BRadshaw 2-8911, Ext. 7114.

### UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

**Man and Civilization: The Control of the Mind.** Saturday through Monday, January 28 through January 30. Twenty-one hours. Fee: $25.00.

**Evening Lecture Series at Richmond Hospital.** Thursday evenings, February 9 through March 16. Fee: $25.00.

**Pain.** Saturday and Sunday, February 11 and 12. Fourteen hours. Fee: $30.00.

**Medicine for General Practitioners.** Mount Zion Hospital. Monday through Friday, February 20 through 24. Thirty-five hours. Fee: $85.00.

**Diseases of the Nervous System in Childhood.** Thursday through Saturday, March 2 through 4. Twenty-one hours. Fee: $150.00.

**Urology.** Thursday and Friday, March 9 and 10. Fourteen hours.*

**Perinatal Problems,** Children's Hospital. Saturday, March 11. Seven hours. Fee: $12.50.

**Diagnostic Radiology.** Wednesday through Sunday, March 15 through 19. Thirty-five hours.*

**Evening Lecture Series in Medicine,** Eden Hospital. Tuesday evenings, April 4 through May 23.†

**Laboratory Investigation of Endocrine Disorders.** Friday through Sunday, April 7 through 9. Twenty-one hours.*

**General Surgery.** Thursday through Saturday, April 13 through 15. Twenty-one hours.*

**Ear-Nose-Throat.** Thursday through Saturday, May 11 through 13. Twenty-one hours.*

**Proctology.** Thursday and Friday, May 18 and 19. Fourteen hours.*

**Water, Salts and Steroids.** Thursday through Saturday, May 25-27. Twenty-one hours.*

**A Course in Ophthalmology.** Thursday through Saturday, June 1-3. Twenty-one hours.*

**A Course in Psychiatry for Physicians in General Practice.** Thursday through Saturday, June 15-17. Twenty-one hours.*

**Cerebral Palsy.** Thursday and Friday, June 29 and 30. Fourteen hours.*

**Fundamental Practices of Radioactivity and the Diagnostic and Therapeutic Uses of Radioisotopes.** Two or three month course limited to one enrollee per month. Fee: $350.00.

For information on courses for physicians or ancillary personnel *contact:* Seymour M. Farber, M.D., assistant dean, Department of Continuing Medical Education, University of California Medical Center, San Francisco 22. MOntrose 4-3600, Ext. 665.

## PRESBYTERIAN MEDICAL CENTER, SAN FRANCISCO

**Common Problems in Pediatrics.** Sunday, January 29. Eight hours. Fee: $25.00.

**Dermatologic Therapy.** Saturday, February 11. Eight hours. Fee: $25.00.

**Diabetes and Thyroid Disease: Current Methods in Diagnosis and Treatment.** Saturday, February 25. Eight hours. Fee: $25.00.

**The Four R's of Fractures: Recognition, Reduction, Retention, Rehabilitation.** Saturday, March 11. Eight hours. Fee: $25.00.

**Problems in Therapy of Cardiac Disease.** Sunday, April 9. Eight hours. Fee: $25.00.

**Problems in Neurology and Neurosurgery.** Saturday, May 6. Eight hours. Fee: $25.00.

**Psychological Problems in General Practice.** Sunday, May 21. Eight hours. Fee: $25.00.

**Horizons in Surgery.** Saturday, June 17. Eight hours. Fee: $25.00.

Note:
Each one of the conferences listed above..... $ 25.00
A series of any 5 conferences..... 100.00

**Operable Heart Disease.** Friday and Saturday, March 3 and 4.

**Conference on Keratoplasty.** Wednesday through Friday, March 8 through 10. Limited enrollment.

**General Review Course for Practicing Physicians.** Thursday through Saturday. March 16 through 18.

**Conference on Strabismus.** Wednesday through Friday. July 12 through 14.

*Contact:* Arthur Selzer, M.D., program committee chairman, Presbyterian Medical Center, Clay and Webster Sts., San Francisco 15, WEst 1-8000, Ext. 303.

*Fee to be announced.

## UNIVERSITY OF SOUTHERN CALIFORNIA, LOS ANGELES

**Nuclear Medicine:**

**Part I,** January. Fee: $50.00.
**Part II,** eight weeks. Fee: $350.00.
**Part III,** twelve weeks. Fee: $350.00.

**Clinical Hematology.** Saturday and Sunday, February 25 and 26.*

**Hawaii Course.** August 2 through 18. The USC School of Medicine will offer the 4th Postgraduate Refresher Course to be held in Honolulu and on board the S.S. Matsonia. (As a time and money saver, air travel is also possible.)

**Cardiac Resuscitation.** Each Wednesday by appointment, 4 to 6 p.m. USC Medical Research Building, Room 211, 2025 Zonal Avenue. Tuition: $30.00. (Each session all-inclusive.)

**Basic Home Course in Electrocardiography.** One year postgraduate series, electrocardiogram interpretation by mail. Physicians may register at any time and receive all 52 issues. Fifty-two weeks. Fee: $100.00.

**Advance Home Course in Electrocardiography.** One year postgraduate series, electrocardiogram interpretation by mail. Fifty-two issues: $85.00. Physicians may register at any time.

*Contact:* Phil R. Manning, M.D., Associate Dean and Director, Postgraduate Division, University of Southern California School of Medicine, 2025 Zonal Avenue, Los Angeles 33. CApital 5-1511.

## COLLEGE OF MEDICAL EVANGELISTS

**SURGICAL ANATOMY (Dissection, Lectures and Demonstrations):**

**Head and Neck.** Monday and Wednesday, April 19 through May 31. Sixty-three hours. Fee: $75.00.

**SURGICAL ANATOMY (Lectures and Demonstrations only):**

**Head and Neck.** Wednesdays, April 12 through May 31. Twenty-four hours. Fee: $35.00.

**Alumni Postgraduate Convention Refresher Courses,** March 12 and 13, on the campus of the College of Medical Evangelists at White Memorial Hospital.

**Joint Manipulation.** Monday through Friday, March 20 through 24. Twenty hours. Fee: $100.00.

**Tropical Public Health.** Monday through Friday, April 3 through 28. Fee: $65.00.

**Clinical Traineeships** available in clinical departments by arrangement with Postgraduate Division and Postgraduate Chairman of department involved. In addition to those listed other traineeships in other departments can be arranged. Eighty hours minimum. Limited enrollment. Begin when individually arranged.

1. **Anesthesia.** Six months. 250 to 300 hours. Fee: $350.00.
2. **Internal Medicine.** Two weeks to nine months.
3. **Pulmonary Diseases** (can be arranged).
4. **Traumatology.** One month. 160 hours. Fee: $125.00.
5. **Urology** (can be arranged).

*For information contact:* Division of Postgraduate Medicine, College of Medical Evangelists, 1720 Brooklyn Ave., Los Angeles 33. ANgelus 9-7241, Ext. 214.

### CALIFORNIA MEDICAL ASSOCIATION ANNUAL SESSION POSTGRADUATE COURSES

**Clinical Neurology** in cooperation with College of Medical Evangelists. White Memorial Hospital. Sunday, April 30, 9-12 a.m.; Monday and Tuesday, May 1 and 2, 2-5 p.m. Fee: $25.00.*

**Office Gynecology** in cooperation with USC School of Medicine. Los Angeles County Hospital. Saturday, April 29, 9-12 a.m. and 2-5 p.m.; Sunday, April 30, 9-12 a.m. Fee: $25.00.*

**Use and Limitations of Laboratory Tests** in cooperation with USC School of Medicine. Los Angeles County Hospital. Saturday, April 29, 9-12 a.m. and 2-5 p.m.; Sunday, April 30, 9-12 a.m. Fee: $25.00.*

**Cardiac Resuscitation:** in cooperation with USC School of Medicine. USC Research Building, Room 211, 2025 Zonal Avenue, Los Angeles 33. Section I: Saturday, April 29, 2-4 p.m.; Section II: Sunday, April 30, 2-4 p.m. (each Section is all-inclusive). Each Section is limited to 10 registrants from areas other than Los Angeles and San Francisco. Fee: $30.00, each Section.*

### CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE CIRCUIT COURSES

**For Dunsmuir, Redding, Chico and Marysville** in cooperation with Stanford University School of Medicine. Begins week of March 5, 1961.

**For Eureka, Ukiah, Napa and Auburn** in cooperation with University of California School of Medicine, San Francisco. Begins week of March 5, 1961.

### CALIFORNIA MEDICAL ASSOCIATION POSTGRADUATE INSTITUTES—1961

**Southern Counties,** February 2 and 3, El Mirador Hotel, Palm Springs, in cooperation with University of Southern California School of Medicine. *Chairman:* Raymond Tatro, M.D., 1875 North "D" Street, San Bernardino.

**West Coast Counties,** March 2 and 3, Del Monte Lodge, Pebble Beach, in cooperation with College of Medical Evangelists. *Chairman:* A. F. Kandlbinder, M.D., 835 Cass Street, Monterey.

**North Coast Counties,** March 23 and 24, Flamingo Hotel, Santa Rosa, in cooperation with University of California, San Francisco. *Chairman:* Milton A. Antipa, M.D., 50 Montgomery Drive, Santa Rosa.

**San Joaquin Valley,** April 14 and 15, Ahwahnee Hotel, Yosemite, in cooperation with UCLA School of Medicine. *Chairman:* J. Malcolm Masten, M.D., 1051 R Street, Fresno.

**Sacramento Valley Counties,** June 29 and 30, in cooperation with Stanford University School of Medicine, Tahoe Tavern, Lake Tahoe. *Chairman:* Joel T. Janvier, M.D., 3632 Marysville Road, Del Paso Heights.

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### AUDIO-DIGEST FOUNDATION

A nonprofit subsidiary of the C.M.A., offers (on a subscription basis) a series of six different hour-long tape recordings covering general practice, surgery, internal medicine, obstetrics and gynecology, pediatrics and anesthesiology. Designed to keep physicians posted on what is new and important in their respective fields, these programs survey current national and international literature of interest and contain selected highlights of on-the-spot recordings of national scientific meetings, panel discussions, symposia, and individual lectures. For information contact Mr. Claron L. Oakley, Editor, 1919 Wilshire Blvd., Los Angeles 57, HUbbard 3-3451.

*Chartered buses for Registrants will leave the Ambassador Hotel one hour before the beginning of each course and will return just after each course is over.

## Medical Dates Bulletin

### JANUARY MEETINGS

MARIN COUNTY HEART ASSOCIATION, Course in Cardiac Resuscitation—3 hour. Saturdays, January 14, 21, 28 and February 4. Marin General Hospital, San Rafael. Fee: $25.00. *Contact:* Richard Hahn, M.D., Chairman, 2044 Fourth St., San Rafael.

LONG BEACH HEART, CANCER AND TB Third Annual Medical Symposium on Diseases of the Heart, Lungs and Chest. January 18, 12:30 p.m., Long Beach Petroleum Club. *Contact:* Leslie R. Raymond, executive director, 2034 Pacific Avenue, Long Beach.

LOS ANGELES TRUDEAU SOCIETY and THE TUBERCULOSIS AND HEALTH ASSOCIATION OF LOS ANGELES COUNTY 8TH BIENNIAL CHEST DISEASE SYMPOSIUM. January 18 through 20. Ambassador Hotel, Los Angeles. *Contact:* Richard S. Gaines, 1670 Beverly Blvd., Los Angeles 26, California.

WESTERN PHARMACOLOGY SOCIETY ANNUAL MEETING. January 22 through 24. Miramar Hotel, Santa Monica. *Contact:* Howard R. Bierman, M.D., President, 9730 Wilshire Blvd., Beverly Hills.

THIRTIETH ANNUAL MID-WINTER CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY. January 23 to 27. Statler-Hilton Hotel, Los Angeles. *Contact:* Norman Jesberg, M.D., treasurer, 500 South Lucas, Los Angeles 17.

WESTERN SOCIETY FOR CLINICAL RESEARCH. January 26 through 28. Carmel-by-the-Sea, Calif. *Contact:* William N. Valentine, M.D., secretary-treasurer, UCLA School of Medicine, Los Angeles 24.

FRESNO COUNTY HEART ASSOCIATION Ninth Annual Central California Cardiovascular Symposium. January 27. 8:30 a.m. to 5:30 p.m. Fresno Elks Club, 5080 E. Kings Canyon Road, Fresno. *Contact:* Jack J. Jacobson, M.D., chairman, Professional Services Committee, 1584 N. Van Ness Ave., Fresno.

### FEBRUARY MEETINGS

CONTRA COSTA COUNTY HEART ASSOCIATION. Co-sponsored by Alameda-Contra Costa Chapter, American Academy of General Practice. 8th Annual Postgraduate Course for Physicians. Eight 2-hour sessions on Monday evening, February 6 through March 27. 8-10 p.m. Concord Community Hospital, 2540 East Street, Concord. *Contact:* Loyse C. Casebolt, Exec. Secretary. 2030 N. Main St., Walnut Creek.

AMERICAN COLLEGE OF PHYSICIANS Southern California Region, Annual Meeting, in cooperation with Northern California and Nevada, Arizona and New Mexico. Biltmore Hotel, Santa Barbara, February 3, 4, 5, 1961. *Contact:* Sherman Mellinkoff, M.D., chairman, scientific program committee, U.C.L.A. Medical Center, Los Angeles 24.

INSTITUTE FOR METABOLIC RESEARCH "Lipid Metabolism in Diabetes and Related Conditions" two-day round table symposium. February 7 and 8. Highland-Alameda County Hospital, Oakland. *Contact:* L. W. Kinsell, M.D., director, Institute for Metabolic Research, 2701 14th Ave., Oakland.

NAPA COUNTY HEART ASSOCIATION 3rd Symposium on Heart Disease. February 15. 10:00 a.m. Recreation Hall, California Veterans Home, Yountville, Calif. *Contact:* Mrs. Zoi Milne, Exec. Secretary, 1121 Brown Street, Napa.

OBSTETRICAL AND GYNECOLOGICAL ASSEMBLY OF SOUTHERN CALIFORNIA, 16th Annual Mid-Winter Clinical Assembly. Ambassador Hotel, Los Angeles, February 13 through 17. *Contact:* Dee Davis, executive secretary, 5478 Wilshire Blvd., Los Angeles 36, WEbster 4-1551.

LOS ANGELES SOCIETY OF NEUROLOGY AND PSYCHIATRY in cooperation with California Spinal Cord Research Foundation, Conference "Recent Contributions of Basic Research to Paraplegia." February 17 and 18. Los Angeles. *Contact:* Robert P. Sedgwick, M.D., secretary-treasurer, 2010 Wilshire Blvd., Los Angeles 57.

CALIFORNIA TUBERCULOSIS AND HEALTH ASSOCIATION, California Trudeau Society Annual Joint Meeting. February 19 through 22, Jack Tar Hotel, San Francisco. *Contact:* Executive director, C.T.H.A., 130 Hayes Street, San Francisco.

SOUTHERN CALIFORNIA SOCIETY OF GASTROENTEROLOGY. "Problems and Pitfalls in Differential Diagnosis of Jaundice"—Leon Schiff, M.D., February 28, Los Angeles County Medical Association. *Contact:* William E. Molle, M.D., secretary-treasurer, 6221 Wilshire Blvd., Los Angeles 48.

## MARCH MEETINGS

SECOND LOW-BEER MEMORIAL LECTURE. University of California School of Medicine. March 2, 8:00 p.m. Auditorium-S, Medical Sciences Bldg., U. C. San Francisco. *Contact:* F. Buschke, M.D., Professor of Radiology, University of California Medical Center, San Francisco 22, Calif.

SOUTHWESTERN PEDIATRIC SOCIETY Postgraduate Lecture Series. March 7 and 8, Statler Hotel, Los Angeles. *Contact:* Harry O. Ryan, M.D., secretary, 194 N. El Molino, Pasadena.

ORANGE COUNTY HEART ASSOCIATION, Annual Symposium on Heart Disease. Saturday, March 11. All day. Charterhouse Hotel, Anaheim. *Contact:* Howard G. Buswell, Exec. Director, P. O. Box 1704, Santa Ana.

ANESTHESIA SECTION OF LOS ANGELES COUNTY MEDICAL ASSOCIATION 6th Annual Spring Postgraduate Meeting. March 11 and 12. Statler Hilton Hotel, Los Angeles. *Contact:* Thomas W. McIntosh, M.D., 686 East Union Street, Pasadena.

COLLEGE OF MEDICAL EVANGELISTS Annual Alumni Postgraduate Convention. Scientific Assembly, Ambassador Hotel, March 14, 15 and 16. *Contact:* F. Harriman Jones, M.D., general chairman, College of Medical Evangelists, 316 North Bailey Street, Los Angeles 33.

PIONEERS MEMORIAL HOSPITAL 11th Annual Medical and Surgical Postgraduate Assembly. March 17-18. Pioneers Memorial Hospital, Brawley. *Contact:* George C. Holleran, M.D., Program Chairman, P. O. Box 159, Brawley.

## APRIL MEETINGS

INDUSTRIAL MEDICAL ASSOCIATION. Biltmore Hotel, Los Angeles, April 11 through 13. *Contact:* Leonard Arling, M.D., secretary, The Northwest Industrial Clinic, 3101 University Avenue, S.E., Minneapolis 14.

CALIFORNIA MEDICAL ASSOCIATION Annual Meeting, Ambassador Hotel, Los Angeles. April 30 through May 3. *Contact:* John Hunton, executive secretary, 693 Sutter Street, San Francisco 2; or Ed Clancy, director of public relations, 2975 Wilshire Blvd., Los Angeles 5.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY ANNUAL MEETING. April 30-May 4. Riviera Hotel, Palm Springs. *Contact:* Al Miller, M.D., Secretary, 500 South Lucas Ave., Los Angeles 17.

## MAY MEETINGS

LOS ANGELES COUNTY HEART ASSOCIATION Annual Meeting, May 3, 12:00 noon to 2:00 p.m. Pacific Ballroom, Statler Hilton Hotel, Los Angeles. *Contact:* Mrs. Sally Smalley, Public Information Director, 2405 W. 8th Street, Los Angeles 57.

HAWAII MEDICAL ASSOCIATION ANNUAL MEETING. May 4-7. Honolulu, Hawaii. *Contact:* Lee McCaslin, Executive Secretary, 510 So. Beretania, Honolulu 13.

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS (for members and invited guests). May 10-12. Del Monte Lodge, Pebble Beach. *Contact:* William J. Engel, M.D., Secretary-Treasurer, Cleveland Clinic, 2020 E. 93rd St., Cleveland 6, Ohio.

NEVADA CHAPTER AMERICAN ACADEMY OF GENERAL PRACTICE. May 18-20, Riverside Hotel, Reno, Nevada. *Contact:* John M. Watson, Secretary, 1845 Prater Way, Sparks, Nevada.

MEDICAL STAFF OF CHILDREN'S HOSPITAL OF THE EAST BAY Ninth Annual Clifford Sweet Seminar. May 18, 19 and 20. Hotel Claremont, Berkeley, and Children's Hospital of the East Bay. *Contact:* Seymour J. Harris, M.D., chairman, Lectureship Committee, 401 29th Street, Oakland 9.

CALIFORNIA HEART ASSOCIATION Annual Meeting and Scientific Session. May 19 through 21, Disneyland Hotel, Anaheim. *Contact:* J. Keith Thwaites, Exec. Director 1370 Mission Street, San Francisco.

AMERICAN ORTHOPAEDIC ASSOCIATION (members and guests). May 22-25. The Ahwahnee Hotel, Yosemite. *Contact:* Lee Ramsay Straub, M.D., Secretary, 535 E. 70th St., New York 21.

AMERICAN UROLOGICAL ASSOCIATION, INC. May 22-25. Biltmore Hotel, Los Angeles. *Contact:* Mr. William P. Didusch, Executive Secretary, 1120 N. Charles St., Baltimore 1.

MEMORIAL HOSPITAL OF LONG BEACH, Third Annual Medical Staff Symposium. May 24. New Memorial Hospital, 2801 Atlantic Ave., Long Beach 6. *Contact:* George X. Trimble, M.D., secretary, Memorial Hospital of Long Beach.

## SUMMER AND FALL MEETINGS

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION Annual Meeting (joint with U. S.-Mexico Border Public Health Association). June 26 through 29. El Cortez Hotel, San Diego. *Contact:* Robert E. Mytinger, M.P.H., director, Executive Office Western Branch, APHA, 693 Sutter Street, San Francisco 2.

NEVADA STATE MEDICAL ASSOCIATION 58th Annual Meeting and 11th Annual Conference of the Reno Surgical Society. August 23-26. Reno, Nevada. *Contact:* Mr. Nelson B. Neff, Exec. Secretary, Nevada State Medical Association, 506 Humboldt St., Reno.

WASHINGTON STATE MEDICAL ASSOCIATION Annual Convention. September 17-20. Olympic Hotel, Seattle, Wash. *Contact:* R. W. Neill, 1309 7th Ave., Seattle.

LOS ANGELES COUNTY HEART ASSOCIATION Professional Symposium. October 11-12. 9 a.m.—5 p.m., Statler Hilton Hotel, Los Angeles. *Contact:* Manuel Siegel, Program Director, 2405 W. 8th St., Los Angeles 57.

CALIFORNIA ACADEMY OF GENERAL PRACTICE 1961 Scientific Assembly. October 15-18. Statler Hilton Hotel, Los Angeles. *Contact:* William W. Rogers, Exec. Secretary, 461 Market Street, San Francisco 5.

# THE PHYSICIAN'S *Bookshelf*

**CARDIAC EMERGENCIES AND RELATED DISORDERS—Their Mechanism, Recognition and Management**—Harold D. Levine, M.D., Senior Associate in Medicine, Peter Bent Brigham Hospital, Boston, Mass.; Assistant Clinical Professor of Medicine, Harvard Medical School. Landsberger Medical Books, Inc., 51 East 42nd Street, New York, N. Y., 1960. 381 pages, $12.00.

This monograph by Dr. Levine is a sound attempt to present a practical account of the treatment of the broad field of cardiac emergencies. It is the outgrowth of seminars presented to senior students over a period of years and therefore is a general clinical account rather than a discussion of the subject in depth or a presentation of recent advances. The author has had some difficulty in separating the emergency from the long-term treatment, at times considering long-term treatment as part of the emergency, such as anticoagulant therapy and in others omitting long-term considerations such as the prevention of recurrent cardiac arrhythmia.

Although an effort has been made to explain the mechanism of the various disturbances, this aspect of the subject has not been considered in depth. Therapy has been discussed in a conventional, practical fashion although at times the author fails to be sufficiently precise in terms of what he recommends as the choice among a variety of potential treatments. For example under paroxysmal atrial tachycardia he uses such phrases as "another choice of medication . . . digitalis . . .," or "the medical literature also reports the use of calcium gluconate," or "one may use various medicinal forms of treatment, probably the simplest and most effective is the subcutaneous injection of 0.5 to 1.0 or even 2.0 mgms. of prostigmine. . . ." The reader is left in doubt as to how the author actually treats the arrhythmia and what he does if the particular treatment that he uses is not effective. It would be most helpful if the author clearly stated the measures which in his own experience were found to be reliable rather than to list all the various methods that have been used.

The discussion of digitalis toxicity is the subject of a special chapter and is most helpful. The reviewer would have liked to have seen a special section on the problem which often presents a real emergency, of when an arrhythmia is the result of excessive digitalis and when it is due to inadequate digitalis. In addition the problem of hypokalemic alkalosis could well be introduced into the discussion of electrolyte disturbances in the chapter on "refractory" congestive heart failure and the distinction between sodium depletion and sodium dilution be more clearly defined. A notable omission in the book concerns emergency treatment of hypertension or a vascular emergency.

In addition to the usual cardiac emergencies, the author has chapters on unusual and noncardiac causes of chest pain, pulmonary embolism, syncope, and cardiac arrest and resuscitation. Each chapter has pertinent references which is a distinct advantage in a monograph of this sort.

The book in general can be recommended as a good account of contemporary thought, but in view of the high price ($12) one wonders whether the reader would not profit more from purchasing one of the standard texts on heart disease.

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**TREATMENT OF CANCER AND ALLIED DISEASES—SECOND EDITION—Tumors of the Breast, Chest and Esophagus, Volume IV**—By Sixty-seven Authors, George T. Pack, M.D., F.A.C.S., and Irving M. Ariel, M.D., F.A.C.S., editors. Paul B. Hoeber, Inc., Medical Division of Harper & Brothers, 49 East Thirty-third Street, New York 16, N. Y., 1960. 667 pages, $30.00.

The precedent created by the first three volumes of the second edition of this nine-part work is ably continued. The excellent format, clear illustrations and legible text of the earlier volumes are maintained.

In a section on tumors of the *breast*, the senior editors reprint the well-known curve of Nathanson and Welch dealing with survival for untreated cases of breast cancer "a natural yardstick for measuring results of therapy." They fail to point out that these survival figures are estimated from the time of recognition or self diagnosis by the patient, and are not comparable with treated results, since the latter usually are and always should be dated from time of definitive treatment. A curve, corrected by the reviewer to allow for the usual delay of about six months in treated cases has been published (American Journal of Roentgenology, December 1954, page 938).

Since the authors then publish several tables and curves dealing with relative survival data from large centers such as the Mayo Clinic, it would be desirable that the next edition carry an absolute survival rate for treated cases of cancer of the breast from a representative group of centers, drawn to scale with the existing one for untreated cases.

The place of so-called radical mastectomy receives understandable emphasis in this worthy surgical text. But it is difficult to understand why the extended supraradical mastectomy (with supraclavicular and mediastinal excision) should merit a whole chapter. Halstead tried and discarded this mutilating procedure more than half a century ago, and it is questionable whether 11 pages of text and 10 beautiful illustrations should be wasted on it.

The place of orthovoltage radiation therapy in the treatment of breast cancer is ably outlined by Stenstrom and Stone. But the senior editors have appended to this a rather futile method of irradiating the internal mammary vessels which cannot be recommended, especially in the light of Haagensen's work showing that when the internal mammary chain is involved, systemic spread has usually occurred.

The valuable work of the modern Edinburgh school headed by a distinguished group of Scottish surgeons and the radiotherapist McWhirter receives scant mention. Since the absolute results by this method of treatment of carcinoma of the breast equal if they do not surpass the comparable results of "radical" mastectomy, the reason for this omission is ob-

scure. Since the recorded death rate for carcinoma of the breast in the United States is increasing, and since radical mastectomy is now regarded by many as merely a good palliative procedure "allowing a maximum number of women to die more comfortably of visceral metastases without local chest wall or axillary recurrence," this reviewer regards the omission of a chapter on the McWhirter program as one meriting correction.

The sections on *thoracic tumors* are introduced with a copy of Rouviere's well known drawing of the lymphatic drainage of the lungs. Since more than 50 per cent of bronchogenic carcinomas originate in the periphery of the lungs, and lie in a bed of numerous thin walled veins, the importance of vascular spread should precede consideration of lymphatic involvement. The poor results attained by any method in the treatment of bronchogenic carcinoma attest this early vascular spread and are considered in subsequent chapters. The authors of the section dealing with bronchial adenomas recommend conservative removal of suitable cases and probably wisely condemn the use of radon seeds in the management of these cases.

It is difficult for the reader to ascertain the senior editor's own preference in the treatment of primary bronchial carcinomas suitable for surgical intervention. There are chapters on "radical lobectomy," pneumonectomy and certain modifications of same. The reader would probably welcome a recommended general policy.

Lampe provides a sound chapter on the radiotherapy of bronchial carcinoma, stressing the usefulness of orthovoltage roentgen therapy and its value in properly selected cases.

The section on tumors of the *esophagus* is the shortest one in the book. An undue amount of space is accorded the radical surgical removal of cancer of the esophagus in the light of the extremely modest results. The by-pass operation is considered in detail. Various methods of radiotherapy of esophageal cancer are discussed, ranging from interstitial radium to betatron treatment. An interesting chart on page 598 shows that the average survival of patients treated with external radiation sources is best with 250 KV. Cobalt and million volt therapy produced a shorter average survival time.

The place of rotation therapy is well discussed by Ebenius and colleagues, and the importance of constant check of the radiated field illustrated by photographs and diagrams. The results are modest, but again better than any published with cobalt or megavoltage, including linear accelerators. Ebenius and colleagues use 200 KV with constant fluoroscope check.

The profession is indebted to Drs. Pack and Ariel for assembling and editing the large amount of valuable material in this volume.

L. H. GARLAND, M.D.

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**EDEMA—Mechanisms and Management—A Hahnemann Symposium on Salt and Water Retention**—Edited by John H. Moyer, M.D., Professor and Chairman of the Department of Medicine, and Morton Fuchs, M.D., Assistant Professor of Medicine, Hahnemann Medical College and Hospital. W. B. Saunders Company, Philadelphia, 1960. 833 pages, $15.00.

This outstanding volume is the second of the symposia held last year at Hahnemann Medical College in Philadelphia, and maintains the high standards of the first. The date of the meeting is not stated in the volume but it is clear from the references that it was last year. The book consists of short articles by 90 authors, comprehensively reviewing the entire subject of edema. Since edema crosses various specialties, investigators from specialties other than internal medicine are included in the symposium.

The book consists of eight parts, beginning with the basic physiology of fluid and electrolyte balance which includes a discussion of cell membrane and electrolyte transfer. A discussion of the newer aspects of the counter current osmotic mechanisms of tubular reabsorption, of hormonal effects on electrolytes, aldosteronism, and the mechanisms of hyponatremia are among the subjects discussed in this important first part. Part II is a basic and clinical discussion of the pharmacology and therapeutic use of diuretics. Emphasis is given to the more recent developments. The coverage in this section is thorough with emphasis on basic mechanisms. The remaining sections cover edema secondary to steroids, electrolytes, diuretics in the treatment of hypertension, toxemia of pregnancy, edema of renal origin, of liver origin, and that associated with congestive heart failure. The articles are of a high order of competence and the references at the end of each article indicate that material of 1958 and 1959 is included. The book is profusely illustrated and many charts and diagrams aid the reader in understanding the highly technical material covered in the book.

This is probably the most comprehensive account of the subject of edema both from the physiological and the clinical aspects and should provide a much greater understanding of the complex problems which present themselves in the treatment of edema. The book can be highly recommended to all physicians interested in understanding the mechanisms and pathophysiology of electrolytes, diuretics and the diseases in which they play an important role.

MAURICE SOKOLOW, M.D.

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**OBSTETRICS (From the Original Text of Joseph B. DeLee, M.D.) Twelfth Edition**—J. P. Greenhill, M.D., F.A.C.S., F.I.C.S. (Hon.) Senior Attending Obstetrician and Gynecologist, The Michael Reese Hospital; Obstetrician and Gynecologist, Associate Staff, The Chicago Lying-in Hospital. W. B. Saunders Company, Philadelphia, 1960. 1098 pages, 1219 illustrations on 903 Figures, 119 in Color, $17.00.

The new edition of this major American textbook appears just five years after the previous edition, thus attesting to its popularity, but it has indeed been extensively rewritten and revised. Nine chapters have been squeezed within a framework of only ten additional pages, and the number of pages devoted to indexing is precisely the same as before. Twenty of the eighty chapters were contributed by twenty-three assisting experts, most of them nonobstetricians, and some eight other consultants reviewed and advised in the preparation of various portions of the text. Practically every page of the book has been rewritten to some extent, a good many of the older illustrations were deleted, and 162 new ones added. In many instances revision meant insertion of new fragments of text, sometimes without serious consideration of precise placement in relation to what preceded and followed. A notable example is in the section on clinical course of labor, where Caldeyro's work is quoted (and several of his graphs reproduced, without credit being given) far ahead of any mention of his name in the text. Then there follows a brief but rather anticlimactic description of some of Caldeyro's earlier work, more or less as stated in the previous edition, although even this paragraph did not escape some degree of editorial revamping. Throughout the book countless minor changes in sentence structure have been made, presumably to substantiate the claim of extensive revision, but most of these accomplished little beyond an increased work load for the typesetter.

Nevertheless, many parts of this book merit the attention of all obstetricians. Particular mention should be made of the stimulating chapter by Ernest Page on placental physiology, Erik Rydberg's explanation of cephalic labor mechanisms, and McCartney's sensible discussion of toxemia.

Davidsohn now receives full credit for the section on erythroblastosis, and this has been suitably brought up to date. Meyer Perlstein, the well-known pediatric neurologist, has contributed a short chapter on perinatal brain injury, with particular emphasis on cerebral palsy, and Honoria Acosta-Sison has expanded the excellent discussion of hydatidiform mole and choriocarcinoma. Helene Deutsch's chapter on the psychology of pregnancy has been replaced by a similar essay written by the late Flanders Dunbar.

Near the end of the book is a very short but rather fascinating chapter by the late Herbert Schmitz. Entitled "Medicomoral Problems," it is a brief discussion of the views of the Catholic Church on such matters as abortion, destructive operations on fetuses, operation for ectopic pregnancy, birth control, and artificial insemination. This should be read by all house officers who have not had occasion to acquaint themselves with Catholic doctrines that often influence obstetric and gynecologic decisions and practices.

Obviously one cannot in this setting review in depth a book of more than a thousand pages covering every facet of obstetrics and related problems, nor is there need to do so for a volume known to all workers in its field. Certainly Greenhill's text is still amongst the top three American efforts, but one gets the impression that to some extent it has gradually taken on the flavor of a reference work rather than the homespun quality of an undergraduate textbook that it had in DeLee's time. Perhaps this is good, even inevitable, as more and more information is added to the hard core of what we used to call obstetrics.

CHARLES E. MCLENNAN, M.D.

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**DISEASES OF THE NEWBORN**—Alexander J. Schaffer, M.D., Associate Professor of Pediatrics, with a section on Neonatal Cardiology by Milton Markowitz, M.D., Assistant Professor of Pediatrics; both of the Johns Hopkins Medical School, and Pediatricians to the Johns Hopkins Hospital. W. B. Saunders Company, West Washington Square, Philadelphia 5, Pa., 1960. 878 pages, illustrated, $20.00.

Doctor Schaffer's extensive experience of almost thirty years of caring for newborns at Sinai Hospital of Baltimore, Wonani Hospital of Maryland and the Johns Hopkins Hospital is well presented by this book. It is a comprehensive and authoritative volume on "Neonatalogy"—a word coined by Doctor Shaffer to describe the art and science of diagnosis and treatment of disorders of the newborn. There are 214 case summaries incorporated into the text to illustrate in a practical and realistic way the facets of differential diagnosis and of treatment. Illustrations are numerous and of excellent quality. References are adequate and up-to-date.

Centers caring for newborns and physicians responsible for their welfare will find this book well worth while.

WILLIAM C. DEAMER, M.D.

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**MOSTLY MURDER** —Sir Sydney Smith, C.B.E., LL.D., M.D. (Edin.), F.R.C.P. (Edin.), Emeritus Professor of Forensic Medicine, Edinburgh University. Formerly Dean of the Faculty of Medicine and Rector of Edinburgh University. With a foreword by Erle Stanley Gardner; David McKay Company, Inc., 119 West 40th Street, New York 18, N. Y., 1960. 318 pages, $4.95.

Sir Sydney Smith, one of the first medico-legal experts of our time, has prepared his autobiography to consist primarily of recounting the events and his role in a number of celebrated criminal cases.

As a pioneer in the field he takes just credit for bringing scientific crime investigation to the fore, particularly in its medical aspects. He simultaneously heaps repeated scorn on his arch rival and colleague Sir Bernard Spilsbury, a pathologist of renown in England. So blunt is this condemnation over Spilsbury's possible errors in cases where both men gave testimony in the courts, that one cannot but wonder that the acrimony in this rivalry would not have waned somewhat after Spilsbury's death. From reading the book, presumably, Sir Sydney Smith did not err at any time.

Despite this, the book presents interesting facets of cases that make fairly satisfying reading. To the person interested in forensic science, the book holds little of technical value that he would not have gathered in other reading. To the casual reader, interested in murder-mystery type material, this book would prove to be worth while, although laborious reading. The book did not prove to be one not easily put down before finishing.

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**LEUKOCYTE ANTIGENS AND ANTIBODIES**—Roy L. Walford, M.D., Assistant Professor of Pathology, University of California School of Medicine, Los Angeles. Grune and Stratton, Inc., 381 Fourth Ave., New York 16, N.Y., 1960. 182 pages, $6.75.

This book is a comprehensive compendium of available information on leukocyte immunology, exclusive of the role of leukocytes in the passive transfer of delayed hypersensitivity and the role of the leukocyte in antibody transport and synthesis.

The author has first-hand experience in practically all features of the problem which he covers, so that his analysis of data is particularly meaningful and critical. Of outstanding value to students and investigators in the field is his detailed description of techniques for the study of leukocyte antigens and antibodies. Included are methods of collection of leukocytes, fractionation into various types, preservation of cells, and immunologic techniques for demonstrating antigen-antibody reactions. The comparative value and meaning of results obtained by various workers using different techniques are assessed, with particular emphasis on the antiglobulin consumption test, which, in the author's opinion, is "the most important technical advance since the development of the leuko-agglutinin test."

The aspect of immuno-hematology covered in this work is among the most controversial and technically difficult. One cannot escape the feeling, after studying the subject, that the true significance of many present concepts of leukocyte immunology will be radically cha ed as methodology progresses. It is refreshing, therefore, to find that the author of this work stresses the errors and pit-falls in the field and avoids formulating broad schemes relating this or that laboratory finding to clinical disease. For example, he points out that the relationship of leukocyte antibodies to leukopenic states is far from clear in that a significant percentage of cases do not demonstrate antibodies, while in some cases where antibodies are present, leukopenia does not exist. One of the few clinical phenomena, that can be unquestionably related, is the high frequency of leukocyte an bodies in patients receiving multiple transfusions. Even here, however, individuals with a high titer of leukocyte antibodies do not always incur the pyrogenic reaction after transfusion which has been attributed to leukocyte antigen-antibody reactions. Also emphasized is the difficulty in establishing the specificity of leukocyte agglutination by serum containing red-cell antibodies, the need for avoiding certain pathological leukocytes and sera as test systems, and the difficulty in interpretation of leukopenic responses *in vivo* as meaning leukocyte antigen-antibody reaction.

A separate chapter on the L-E phenomenon is of considerable interest since it draws together the present thinking on the meaning of the phenomenon. It is clear that this activity involving nuclear damage and phagocytosis is distinct from the leukocyte antigen-antibody reactions as de-

tected by leukoagglutination, etc. Another chapter deals with the important question of the relationship between humoral leukocyte antibodies and the homograft reaction. The evidence *pro* and *con* is well covered with no definite conclusion being reached.

This book will be of great value as a reference point for all workers dealing with leukocyte immunology. Although it does not cover the field completely, it is the best summarization, to date, of the highly specialized study of leukocyte antigen-antibody reactions.

CHARLES G. CRADDOCK, M.D.

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**RADIATION—Use and control in Industrial Application**—Charles Wesley Shilling, M.D., Sc.D., Deputy Director, Division of Biology and Medicine, U. S. Atomic Energy Commission, Washington, D. C. Grune & Stratton, 381 Fourth Ave., New York 16, N. Y., 1960. 223 pages including 13 pages of appendices and 10 columns of index, $6.75.

The author is Deputy Director of the Division of Biology and Medicine of the U. S. Atomic Energy Commission. In this book he attempts to do his duty in necessary indoctrination of the many industrial physicians who will be responsible for radiation hygiene in the rapidly expanding uses of radiation and nuclear reactions. He has managed to get most of the important things into this small book. He has shown admirable restraint in telling the physical foundations, which most authors find so romantic and so satisfying in our relatively exact knowledge of it that they give way to temptation and tell it all over again. He mentions briefly the sources of radiation hazard with reasonable evaluation of their importance. He gives a round picture of biological effects—the acute syndrome, the delayed effects and sequelae and the genetic hazards; with a really brief, well pointed sketch of genetic mechanisms.

A very short chapter on radiation accidents is followed by short notes on 21 accidents and incidents. Control of exposure, licensing, monitoring, barriers and therapy are discussed briefly. The chapters on radiation safety and the work of the health physicist and the role of legislation are a bit fuller.

Instruments and techniques of measurement are handled very briefly indeed, as are the problems of decontamination. No attempt was made to produce a technical handbook.

The disposal of radioactive waste is seen to loom very large, especially from nuclear power. The rapidly expanding uses of radioactive isotopes for control and research are illustrated with some examples. There'll be plenty of problems.

The author mentions nearly everything that should have the attention of an industrial physician. He gives quantitative values where need or curiosity can be anticipated. I find very many small things to quarrel with, yet on second look find most of them only a matter of what aspect is emphasized.

In a few places the author attempts to wrap up in a sentence what would require a goodly paragraph to state fairly in our present state of knowledge and controversy. Examples are relative effects of gamma, beta, alpha and neutron rays and the oxygen effect. In one chapter on basic effects the attenuation by distance and by absorption according to thickness are confused. In relating the effect of accidental large exposures part of the dose is given in r of 80 kv x-rays. This must have been a calculated biologic equivalent of estimated neutron dose, but is not so stated. The damaging fetal dose mentioned is 200r, whereas it is well known that 25r may be effective. The probable statistical fallacy in the reported 5 year life shortening among U. S. radiologists is not cited. A serious typographical error reverses the intended statement about a threshold dose in leukemia induction.

Some statements about protection will be seen by any medical radiologist as unwarranted. Examples: "Every x-ray unit . . . or . . . located underground, in the side of a hill . . ."; ". . . in fluoroscopy, the x-ray tube target should be at least 18 inches from . . . the patient"; ". . . bordering on malpractice is the so-called pediatric 'preventive' fluoroscopic examination of young infants." This last is likely to make some pediatricians' hair stand on end. I think the author overly hopeful about chemical therapeutic agents for radiation injury. He attempts to give the relative effectiveness of lead, concrete and steel all in one statement. He'd better not have tried.

Like everyone else connected with AEC, he calls uranium and plutonium "special nuclear material." Among his general radiologic safety rules appears: "9. All experiments using one microcurie . . . will be conducted in hoods or glove boxes." I wonder if the author would faint to see 150 millicuries of Au-198 injected into a patient's abdomen as she lies in bed.

There is one paragraph that I wish I could rewrite—the one on Psychology and Publicity Problems. In case of an accident "The newspapers will get the information first from 'tips' and will then call the physician to get the 'gory' details. The only safe answer is, 'No comment, call the public relations officer.'" I do think that we ought to be as informative and helpful to newspaper reporters as we can. I also think that the ideal industrial physician makes the ideal public relations officer.

Having combed out all these defects, I'd like to epitomize this book as a valuable view of nearly the whole of an industrial physician's obligatory information about radiation hazards in a space small enough that he will probably read all of it.

R. R. NEWELL, M.D.

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**SHAW'S TEXTBOOK OF OPERATIVE GYNAECOLOGY—Second Edition**—Revised by John Howkins, M.D., M.S. (London), F.R.C.S. (Eng.), F.R.C.O.G., St. Bartholomew's Hospital, University of London and University of Cambridge. The Williams & Wilkins Company, Baltimore, 1960. 484 pages, $20.00.

In the preface to this second edition Howkins, gynecologist at St. Bartholomew's Hospital, says he has undertaken the task of revision as a gesture of gratitude to his former chief, who at his death bequeathed to him the editorship of future editions. Some chapters have been extensively revised and a few new ones added. It was the editor's purpose to describe only those operative techniques which he and his colleagues consider to be most acceptable and which they have personally found to be useful.

After several introductory chapters dealing rather sketchily with pre-operative study and preparation, anesthesia, and postoperative problems, the major portion of the book is devoted to well illustrated descriptions of all the common gynecological operations and a few uncommon ones. Two short chapters are concerned with operative injuries of bladder, ureter and intestine, as well as the more frequent intestinal lesions encountered in the course of gynecologic surgery. Cesarean section is considered briefly, and there is a chapter by I. G. Williams, a radiotherapist, on radiation for carcinoma of the cervix, endometrium, vagina and ovary. The final page of the text contains directions for the surgical management of cardiac arrest.

This book is a blending of textbook and surgical atlas, but certainly its usefulness in this country will be largely as an atlas. The illustrations by Leslie Caswell are superb examples of the medical illustrator's art and may be recommended to all residents in gynecology and to others interested in the technical details of gynecologic surgery.

CHAS. E. MCLENNAN, M.D.

**EMERGENCY SYNDROMES IN PEDIATRIC PRACTICE, THE**—Alfred J. Vignec, M.D., Clinical Professor of Pediatrics, New York University, College of Medicine, New York, N. Y.; Medical Director and Pediatrician in Chief, New York Foundling Hospital, New York, N. Y.; and Director of Pediatric Division, St. Vincent's Hospital, New York, N. Y. Landsberger Medical Books, Inc., 51 East 42nd Street, New York, N. Y., 1959. 382 pages, $9.00.

This book is intended for the practicing physician who in his day to day practice encounters problems in infants and children that require immediate attention. The author recognizes the dilemma that is presented when one has to treat while at the same time trying to establish the diagnosis. He directs his attention to both phases of such a situation.

There are chapters on the newborn, respiratory tract, cardiovascular system, the gastrointestinal tract, hematopoietic system, central nervous system, metabolic disorders, the use of steroids, poisoning, the premature and allergy. The covering of the acute emergency situation in each of these sub-divisions is thorough, readable and to the point, albeit at times controversial. The illustrations employed are entirely those of roentgenograms and are unfortunately of poor quality. The volume is recommended for those who see a large number of acutely ill infants and children.

WILLIAM C. DEAMER, M.D.

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**MEDICAL LICENSURE EXAMINATIONS (Rypin's)—Topical Summaries and Questions—9th Edition**—Walter L. Bierring, M.D., M.A.C.P., M.R.C.P., Edin. (Hon.), Director, Division Gerontology, Heart and Chronic Diseases, Iowa State Department of Health; Secretary-editor Federation of State Medical Boards of United States; Professor of Medicine Emeritus, State University of Iowa, College of Medicine; and former Iowa State Commissioner of Health. With the Collaboration of a Review Panel. J. B. Lippincott Company, East Washington Square, Philadelphia 5, Pa., 1960. 805 pages, $11.00.

The ninth edition of Rypin's Medical Licensure Examinations has been "thoroughly revised and updated" since its last edition in 1957. The text is in two principal sections, Part I, the basic medical sciences and Part II, the clinical sciences. It is evident on reviewing the book that "every effort has been made to treat as concisely as possible those portions of the medical curriculum generally selected for use by the various examining boards."

The introductory chapter describes the methods used now in the examinations and gives some examples of the objective multiple choice written tests as well as examples of questions incorporating a patient problem.

Because of the vast amount of information that has been crammed into a total of 787 pages, there is naturally limitation in what can be presented on each topic, but even though many are discussed in outline form only, much is brought to the attention of the reader for further study if necessary.

There are a few areas in the book where some additional information or a reference to an authoritative work or textbook would seem desirable in order to help the student's grasp of the subject. For example, in the discussion on the respiratory system, it undoubtedly would be of value to have the normal pulmonary function tests tabulated along with results of tests of a few pathological conditions such as emphysema or fibrosis of the lungs. Or, in the part on cardiac surgery, a reference to some special articles giving the most recent experiences with this pioneering work would help the student. The few lines on electrocardiography are too brief to be of much help.

The chapter on psychiatry is very good and has a short, excellent list of references. This is the only chapter in the text that provides such supplementary information.

These criticisms are meant to be constructive suggestions and are in no way critical of the excellent manner in which the majority of topics are discussed. Examples of information concisely provided are the paragraphs on blood types, on leukemias, and on antibiotics.

In conclusion it can be said that this volume provides excellent up-to-date information on all divisions of medical science and clinical medicines; that it might in some way strengthen a few topics that are of special interest today; but the information that has been condensed in this text will serve the student well who is preparing for examinations for licensure and is an excellent review for any physician.

HENRY GIBBONS, III, M.D.

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**INJURIES OF THE BRAIN AND SPINAL CORD AND THEIR COVERINGS — 4th Edition** — edited by Samuel Brock, New York University. Springer Publishing Company, Inc., 44 East 23rd St., New York 10, N.Y., 1960. 739 pages, $18.50.

This volume on injuries of the brain and spinal cord and their coverings edited by Samuel Brock is the 4th edition since the original edition that appeared 20 years ago.

Chapters have been contributed to by 31 different authors, all of whom have national and international prominence.

In my opinion this book would be of great value to every medical library and particularly to the neurosurgeon, the neurologist, the general practitioner, the internist, and the orthopedist.

Of particular interest is the chapter on simulation (malingering) by Dr. Keschner. And also the chapter on the electro-encephalogram in case of a head injury by Dr. Hoefer.

Every phase of injury regarding this subject has been adequately covered. In my opinion, I would regard this as the leading volume on trauma of the nervous system.

ROBERT F. PALMER, M.D.

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**PHYSICIAN'S HANDBOOK—11th Edition**—Marcus A. Krupp, M.D., Associate Clinical Professor of Medicine, Stanford University School of Medicine, Stanford; Director, Palo Alto Medical Research Foundation, Palo Alto; Norman J. Sweet, M.D., Associate Professor of Medicine, University of California School of Medicine, San Francisco; Ernest Jawetz, Ph.D., M.D., Professor of Microbiology and Lecturer in Medicine and Pediatrics, University of California School of Medicine, San Francisco; and Charles D. Armstrong, M.D., Assistant Clinical Professor of Medicine, Stanford University School of Medicine, Stanford. Lange Medical Publications, Los Altos, Calif., 1960. 547 pages, $3.50.

The Physician's Handbook is essentially a compilation of those diagnostic and therapeutic facts and procedures which the authors feel to be of greatest daily interest and value to the student and practitioner of medicine. For this, its eleventh edition, it has been thoroughly revised and reorganized. There are two new chapters on emergency medical examination and chemical analysis of blood and body fluids.

By title and by what it contains this is literally a handbook—handy to have, to carry, to use. It tells the student or practitioner what to do and how to do it. Its 547 pages and the covers too are jam-packed with handy, useful information. As an inexpensive paper-back, kept remarkably well up to date, for the house officer's pocket or the physician's bag this reviewer does not know of a superior volume.

EDGAR WAYBURN, M.D.